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A STATISTICAL ANALYSIS OF PERSONS CERTIFIED BLIND IN SCOTLAND*

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A STATISTICAL analysis of 3,219 persons certified blind at the Regional Clinic for Glasgow and South-West Scotland was published in the *Brit. Jl. of Ophthal.* in 1942 by John Marshall and H. E. Seiler[1].

The present analysis relates to the rest of Scotland, comprising the regional clinics for Edinburgh, Dundee, Dumfries, Aberdeen and Inverness for the period November, 1930, to the end of 1938. During that period a total of 5,953 persons were examined by ophthalmologists and 4,078 or 68·5 per cent. were certified blind within the meaning of the Blind Persons Act.

The definition of blindness is the same for the whole of Scotland and is based on visual standards. The standard is divided into 3 categories, *viz* :—

Group I. Visual acuity of less than 3/60 Snellen. Regarded as blind.

Group II. Visual acuity of 6/60 Snellen or better. Not regarded as blind unless the field of vision is contracted to 10 degrees or less throughout the greater part of its extent and particularly in the lower part of the field.

* Received for publication, January, 1946.

Group III. Intermediate degrees of visual acuity, *i.e.*, 3/60 Snellen but less than 6/60. Regarded as blind—(*a*) When the visual defect is associated with a restricted field of vision although it exceeds the limit of 10 degrees laid down in Group II. (*b*) If aphakic.

The form of medical certificate recommended by the Department of Health for Scotland is used at all regional clinics, and all cases were examined by two ophthalmologists.

A scrutiny of the certificates of blindness was made to ensure that the diagnosis and causes of eye defects found had been entered in accordance with the nomenclature on the certificate, and to verify as far as possible the age of onset of eye trouble and the age of onset of blindness.

This scrutiny revealed that the Wassermann test for syphilis had been employed in selected cases only, and it has therefore been excluded from the tables. The family history has also been omitted as the records were too scanty to be of any statistical value. The recording of different examiners was made to a uniform standard and the code numbers were transcribed from the certificates on to sheets in preparation for machine card recording. The preliminary survey to this point was completed in 1939 but owing to the war it was not found possible to proceed until now. The subsequent compilation of the statistical tables has been carried out on Powers machines by the Department of Health for Scotland.

The number of certified blind persons dealt with in the present investigation reinforced by Marshall and Seiler's figures is 7,297 but this does not cover the whole population, and it is difficult to obtain authoritative information on the total extent of blindness in the country.

Enquiry has been made of the Associations for Teaching the Blind and the consensus of opinion seems to be that about 2 per cent. of blind persons belong to the higher social groups and have not applied for pension or other form of special assistance.

On March 31, 1935, there were 8,332 blind persons on the registers of the local authorities, and the discrepancy is attributed to the following difficulties experienced in the scrutiny of medical certificates.

A large number of certificates related to persons who were not blind. Re-examination forms were commonly not attached to the original certificates and if blindness supervened there was not sufficient information on the re-examination form for classification. In a considerable number of the earlier certificates the information was so meagre that they could not be included in the survey. A further consideration of importance regarding the total number of the blind is the considerable lag in registering blindness which frequently occurs, *e.g.*, Blindness is recorded for 420 persons in the

age group 0—under 1, but only one case was registered as blind at that age.

This lag occurs up to the age group 16-29 and below this group few people appear to register at the onset of blindness.

Out of the total of 4,078 for the rest of Scotland, 612 cases stated they were blind before the age of 16 years, but blindness at ages under 16 on the register amounts to only slightly over 100 cases.

The 50-64 group shows the number of persons blind and on the register as about the same as the number who stated they became blind in this age range, and nearly all the lag is made up in the age group 65 and over.

Speculation about the causes of this lag in registration suggests that while there may be an element of exaggeration in the claims as to the date of onset of blindness this is not the main cause of the apparent deferment in registration. Economic considerations no doubt largely explain the lag because many blind persons only register to obtain the monetary benefits derived after registering when a worsening of their financial circumstances or deterioration in health make this course necessary. It is not without significance that the lag in registration is only made up when pensionable ages are reached. It is thought therefore that the tables showing the alleged age at onset of blindness provide the best clue to the number of persons actually blind at these ages.

It seems that roughly one in eight blind persons do not register at the time of blindness, and in estimating the total blindness in Scotland fully 1,000 would have to be added to the 8,332 on the registers. It may be assumed therefore that the total number of blind persons between the years 1932 and 1939, including the higher social groups, is between 9,500 and 10,000.

Assuming the same incidence of blindness in the populations of England, Wales and Northern Ireland and without making any allowance for the different age and sex constitutions of these populations compared with Scotland, the number of blind persons in the United Kingdom may be estimated approximately at between 80,000 and 90,000.

A recent estimation by Sorsby for England and Wales puts the total at not less than 80,000 and unlikely to exceed 100,000[2].

A point of importance bearing on the accuracy of the tables as reflecting the age distribution of blindness in Scotland is the period covered by the investigation which represents about 8 years. During that time new entrants to the register were brought into the review but persons going off the register were not included. Since death is the main reason for ceasing to be a registered blind person it will be obvious that an influence selective of the younger ages has been operating. To an extent, which, however, it is impossible to assess, certain tables such as that showing the

distribution of present ages, will be more seriously affected than others. Though the data presented here do not therefore represent an accurate cross section of the blind population at any one time most of the tables are relatively unaffected by the age selective element in sampling, and it is considered that they may be used to show the relative importance of age, cause and condition in the blind state. Guidance of the future course of recording and analyses answering questions bearing on the prevention and treatment of blindness are also unaffected.

The chief value of a statistical analysis of the blind is the insight it gives into the study of prevention of blindness, and for this purpose records of monocular blindness would be of great importance. On practical grounds it is appreciated that difficulties exist in making blindness, whether monocular or total, a notifiable disease. Information on the extent of monocular blindness, particularly of the traumatic type whether industrial or otherwise, might be available from the certificates of incapacity for work granted to insured persons and contained in the records of approved societies. An attempt is being made to obtain information from that source.

Marshall and Seiler have discussed very fully the results of their analysis of the cases covered by their investigation, and the figures for the rest of Scotland confirm largely what has been brought out by them. To avoid repetition only comments are given on the tables now published and an explanation of some of the differences in the figures has been suggested.

In order to simplify comparison of the results of this analysis with the figures for Glasgow and South-West Scotland, reference in parenthesis is given at the head of each table to the relevant figures in the Glasgow report.

The aim has been to present an analysis for Scotland by supplementing that already made by Marshall and Seiler for Glasgow and the South-West. In the absence of data always comparable between the two investigations it has not been possible in every case to keep the tables published now for the rest of Scotland in line with those for Glasgow and the South-West. This object, however, has been kept in view and certain tables have been amalgamated to present now for the first time as complete a record as possible of the blind position in Scotland.

It would seem that the difficulties mentioned could be overcome by the adoption of a simple scheme based on the records of local authorities.

A central register was kept in the Department of Health until about the year 1929. If this register were re-established the Statutory Department might in future publish annual statistics showing the number of registered blind at different ages, the cause

TABLE 2 (VI, VII B APPENDIX

CAUSES OF BLINDNESS. SECOND EYE AT PRESENT AGE

CAUSES	TOTALS			CASES BY AGE GROUPS															
	Total	Total	Total	Under 1		1–4		5–15		16–29		30–39		40–49		50–64		65+	
	M. & F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
CONGENITAL AND UNDETERMINED																			
Congenital anomalies	235	128	107	—	—	6	6	22	14	41	25	28	12	10	14	15	28	6	8
Abiotrophies, etc. (Retinitis Pigmentosa) (Leber) (Tay Sachs)	213	137	76	—	—	1	—	8	3	15	13	15	9	29	10	55	31	14	10
Tumour of Globe and Orbit	4	3	1	—	—	1	—	—	—	—	1	—	—	1	—	1	—	—	—
Myopia	563	220	343	—	—	—	—	2	—	22	14	9	17	35	25	93	142	59	145
Other errors of refraction	4	3	1	—	—	—	—	—	—	—	1	1	—	—	—	—	—	2	—
Glaucoma, primary	314	161	153	—	—	—	—	—	—	2	—	3	2	6	7	41	44	109	100
Cataract, primary	706	271	435	—	—	—	—	—	—	4	—	—	3	7	6	70	66	190	360
Other primary ocular defects (Primary Detachment)	54	33	21	—	—	—	—	2	—	1	—	3	1	6	2	13	12	8	6
INFECTIOUS AND TOXIC																			
Exogenous: Ophthalmia Neonatorum	140	70	60	—	—	6	2	4	1	14	12	11	13	13	19	12	18	10	5
Exogenous: Trachoma	35	17	18	—	—	—	—	—	—	1	—	2	2	2	2	3	9	9	5
Exogenous: Local septic infection of coats of eye	153	71	82	—	—	—	—	—	2	1	3	2	3	6	8	30	33	32	33
Exogenous: Other local specific infections (Gonorrhoea) (Mycosis)	5	1	4	—	—	—	—	—	—	—	—	—	—	1	—	—	2	—	2
Endogenous: Gonorrhoea	3	3	—	—	—	—	—	—	—	—	—	—	—	2	—	1	—	—	—
Endogenous: Syphilis, congenital	231	103	128	—	—	1	1	4	5	27	20	12	33	23	23	28	41	8	5
Endogenous: Syphilis, acquired including not definitely congenital	151	103	48	—	—	—	—	—	—	2	2	7	3	21	9	53	24	20	10
Endogenous: Specific fevers (Smallpox) (Measles) (Scarlet) (Diphtheria)	37	20	17	—	—	—	1	—	—	4	3	5	1	3	2	4	5	4	5
Endogenous: Meningitis (non-tuberculous) including Cerebro-Spinal Fever	12	4	8	—	—	—	1	1	2	1	2	1	1	1	2	—	—	—	—
Endogenous: Tuberculosis	24	13	11	—	—	—	—	—	—	3	1	3	2	2	5	2	1	3	2
Endogenous: Phlyctenular, Strumous and similar, not definitely tuberculous	57	23	34	—	—	—	—	2	—	2	3	1	1	4	2	6	14	8	14
Endogenous: Septicaemia, acute	5	3	2	—	—	—	—	—	—	—	—	2	—	—	—	—	2	1	—
Endogenous: Septicaemia, chronic, auto-toxic-focal sepsis	445	190	255	—	—	1	1	1	—	7	8	6	13	34	27	74	103	67	103
Endogenous: Other general infections and organismal diseases	13	6	7	—	—	—	—	—	—	1	1	—	—	1	1	2	2	2	3
TRAUMATIC AND CHEMICAL																			
Birth Trauma	5	3	2	—	—	—	1	1	1	1	—	1	—	—	—	—	—	—	—
Non-industrial Trauma	37	23	14	—	—	—	1	1	—	5	1	5	—	1	3	5	4	6	5
Industrial Trauma	95	92	3	—	—	—	—	—	—	6	—	8	1	12	—	41	—	25	2
War Trauma	25	25	—	—	—	—	—	—	—	1	—	4	—	12	—	7	—	1	—
Trauma, category not ascertainable	2	2	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	1	—
Sympathetic Ophthalmia	57	42	15	—	—	—	—	2	—	3	3	7	3	2	—	16	8	12	1
Chemico-toxic, non-industrial (Tobacco) (Alcohol) (Lead)	11	11	—	—	—	—	—	—	—	—	—	—	—	—	—	6	—	5	—
SYSTEMIC DISEASE																			
Anaemia and Blood Diseases	7	2	5	—	—	—	—	—	—	—	1	—	—	—	1	1	1	1	2
Diabetes	50	15	35	—	—	—	—	—	—	—	—	1	—	1	—	6	23	7	12
Nephritis	9	5	4	—	—	—	—	—	—	—	—	—	—	—	1	5	3	—	—
Pregnancy, diseases of pregnancy or Child Birth (i.e. Eclampsia)	5	—	5	—	—	—	—	—	—	—	1	—	3	—	1	—	—	—	—
Vascular diseases, including cerebral vascular lesions	163	97	66	—	—	—	—	—	—	2	—	2	2	3	—	22	16	68	48
Intracranial Neoplasm	29	12	17	—	—	—	—	4	1	5	—	—	5	—	5	3	4	—	2
Other diseases of central nervous system	33	17	16	—	—	1	1	1	1	—	3	4	4	4	2	6	3	1	2
Functional disturbances (Hysteria) (malingering)	7	4	3	—	—	—	—	—	—	—	—	—	—	—	—	3	1	1	2
Other general diseases	15	9	6	—	—	—	—	—	—	1	—	—	2	2	1	1	2	5	1
Not classified owing to lack of data	124	73	51	—	—	—	1	2	2	7	2	8	7	11	10	24	16	21	13
Totals	4078	2015	2063	—	—	17	16	57	32	179	120	152	143	255	188	649	658	706	906
						33		89		299		295		443		1307		1612	

TABLE 3 (APPENDI

CAUSES OF BLINDNESS. SECOND EYE AG

CAUSES	TOTALS					
	Total M. & F.	Total M.	Total F.	Under 1 M.	Under 1 F.	M.
CONGENITAL AND UNDETERMINED						
Congenital anomalies	235	128	107	58	60	8
Abiotrophies (Retinitis Pigmentosa) (Leber) (Tay Sachs)	213	137	76	7	5	1
Tumour of Globe and Orbit	4	3	1	—	—	2
Myopia	563	220	343	4	2	2
Other errors of refraction	4	3	1	—	—	—
Glaucoma, primary	314	161	153	—	—	—
Cataract, primary	706	271	435	1	1	—
Other primary ocular defects (Primary Detachment)	54	33	21	—	—	—
INFECTIOUS AND TOXIC						
Exogenous: Ophthalmia Neonatorum	140	70	70	42	47	4
Exogenous: Trachoma	35	17	18	—	—	—
Exogenous: Local septic infection of coats of eye	153	71	82	1	1	6
Exogenous: Other local specific infections (Gonorrhoea) (Mycosis)	5	1	4	—	—	—
Endogenous: Gonorrhoea	3	3	—	—	—	—
Endogenous: Syphilis, congenital	231	103	128	10	9	2
Endogenous: Syphilis, acquired, including not definitely congenital	151	103	48	—	—	—
Endogenous: Specific Fevers (Smallpox) (Measles) (Scarlet) (Diphtheria)	37	20	17	—	—	3
Endogenous: Meningitis (non tuberculous) including cerebro-spinal Fever	12	4	8	—	—	2
Endogenous: Tuberculosis	24	13	11	1	—	—
Endogenous: Phlyctenular, Strumous and similar, not definitely tuberculous	57	23	34	—	—	2
Endogenous: Septicaemia, acute	5	3	2	—	—	—
Endogenous: Septicaemia, chronic, auto-toxic, focal sepsis	445	190	255	1	4	5
Endogenous: Other general infections and organismal diseases	13	6	7	—	—	1
TRAUMATIC AND CHEMICAL						
Birth Trauma	5	3	2	1	1	1
Non-industrial Trauma	37	23	14	—	1	—
Industrial Trauma	95	92	3	—	—	—
War Trauma	25	25	—	—	—	—
Trauma, category not ascertainable	2	2	—	—	—	—
Sympathetic Ophthalmia	57	42	15	—	—	2
Chemico-toxic, Non-industrial (Tobacco) (Alcohol) (Lead)	11	11	—	—	—	—
SYSTEMIC DISEASE						
Anaemia and Blood Diseases	7	2	5	—	—	—
Diabetes	50	15	35	—	—	—
Nephritis	9	5	4	—	—	—
Pregnancy, diseases of pregnancy or Child Birth, i.e. Eclampsia	5	—	5	—	—	—
Vascular diseases, including cerebral vascular lesions	163	97	66	—	—	—
Intracranial neoplasm	29	12	17	—	—	2
Other diseases of central nervous system	33	17	16	2	—	—
Functional disturbances (Hysteria) (Malingering)	7	4	3	—	1	—
Other general diseases	15	9	6	—	—	—
Not classified owing to lack of data	124	73	51	2	2	4
Totals	4078	2015	2063	130	134	47
				264		

(APPENDIX E)

EYE AGE AT ONSET OF BLINDNESS

CASES BY AGE GROUPS

nder 1	1–4		5–15		16–29		30–39		40–49		50–64		65+	
F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
60	8	4	16	9	23	12	5	4	9	11	7	6	2	1
5	1	3	15	7	32	15	22	8	29	13	29	20	2	5
—	2	1	—	—	—	—	—	—	—	—	1	—	—	—
2	2	2	5	3	24	21	19	20	43	49	95	184	28	62
—	—	—	—	—	1	1	—	—	—	—	1	—	1	—
—	—	—	—	1	5	3	6	6	6	13	55	54	89	76
1	—	—	2	—	5	—	3	4	12	15	90	115	158	300
—	—	—	3	—	7	2	4	2	5	5	9	10	5	2
47	4	9	8	5	2	1	4	—	5	2	3	5	2	1
—	—	1	1	1	1	2	3	—	2	3	5	7	5	4
1	6	3	4	4	4	5	2	4	11	16	25	31	18	18
—	—	—	1	—	—	—	—	—	—	2	—	—	—	2
—	—	—	1	—	—	—	1	—	—	—	1	—	—	—
9	2	10	26	30	23	31	14	11	15	18	11	18	2	1
—	—	—	—	—	5	5	20	8	22	13	45	20	11	2
—	3	8	7	1	6	1	1	1	1	1	2	4	—	1
—	2	3	—	4	2	—	—	1	—	—	—	—	—	—
—	—	—	2	—	4	4	2	2	1	2	2	3	1	—
—	2	—	5	3	4	6	1	2	4	7	4	10	3	6
—	—	—	1	—	—	—	1	—	—	—	1	2	—	—
4	5	3	15	9	17	33	23	29	28	38	67	85	34	54
—	1	—	1	1	1	1	—	—	1	1	1	2	1	2
1	1	1	1	—	—	—	—	—	—	—	—	—	—	—
1	—	—	8	1	5	1	1	3	2	2	5	4	2	2
—	—	—	1	—	27	—	17	1	15	1	20	—	12	1
—	—	—	1	—	16	—	4	—	3	—	1	—	—	—
—	—	—	1	—	—	—	—	—	—	—	—	—	1	—
—	2	—	11	6	13	3	5	3	2	—	4	3	5	—
—	—	—	—	—	—	—	1	—	1	—	6	—	3	—
—	—	—	—	1	—	1	—	—	—	1	1	—	1	2
—	—	—	—	—	—	—	1	—	1	1	8	24	5	10
—	—	—	—	—	—	—	—	1	1	1	4	2	—	—
—	—	—	—	—	—	1	—	3	—	1	—	—	—	—
—	—	—	—	—	2	1	2	2	3	2	35	20	55	41
—	2	—	4	1	3	2	—	5	2	8	1	1	—	—
—	—	1	—	5	2	3	3	3	4	—	5	3	1	1
1	—	—	—	—	1	—	—	—	1	—	1	1	1	1
—	—	—	2	1	—	1	1	1	1	1	—	1	5	1
2	4	5	6	6	10	8	8	6	13	7	17	10	13	7
134	47	54	148	99	245	164	174	130	243	234	562	645	466	603
264	101		247		409		304		477		1207		1069	

TABLE 4 (

CAUSES OF BLINDNESS. SECOND E

CAUSES		TOTALS			Un
		Total M. & F.	Total M.	Total F.	M.
CONGENITAL AND UNDETERMINED					
Congenital anomalies		235	128	107	108
Abiotrophies, etc. (Retinitis Pigmentosa) (Leber) (Tay Sachs)		213	137	76	35
Tumour of Globe and Orbit		4	3	1	1
Myopia		563	220	343	24
Others errors of refraction		4	3	1	—
Glaucoma, primary		314	161	153	1
Cataract, primary		706	271	435	3
Other primary ocular defects (Primary Detachment)		54	33	21	—
INFECTIOUS AND TOXIC					
Exogenous	Ophthalmia Neonatorum	140	70	70	70
Exogenous	Trachoma	35	17	18	3
Exogenous	Local septic infection of coats of eye	153	71	82	6
Exogenous	Other local specific infections (Gonorrhoea) (Mycosis)	5	1	4	1
Endogenous	Gonorrhoea	3	3	—	—
Endogenous	Syphilis, congenital	231	103	128	19
Endogenous	Syphilis, acquired including not definitely congenital	151	103	48	1
Endogenous	Specific fevers (Smallpox) (Measles) (Scarlet) (Diphtheria)	37	20	17	2
Endogenous	Meningitis (non tuberculous) including Cerebro-Spinal Fever	12	4	8	—
Endogenous	Tuberculosis	24	13	11	1
Endogenous	Phlyctenular, Strumous and similar, not definitely tuberculous	57	23	34	3
Endogenous	Septicaemia, acute	5	3	2	1
Endogenous	Septicaemia, chronic, auto-toxic, focal sepsis	445	190	255	2
Endogenous	Other general infections and organismal diseases	13	6	7	—
TRAUMATIC AND CHEMICAL					
Birth Trauma		5	3	2	3
Non-industrial Trauma		37	23	14	1
Industrial Trauma		95	92	3	—
War Trauma		25	25	—	—
Trauma, category not ascertainable		2	2	—	—
Sympathetic Ophthalmia		57	42	15	—
Chemico-toxic, non-industrial (Tobacco) (Alcohol) (Lead)		11	11	—	—
SYSTEMIC DISEASE					
Anaemia and Blood Diseases		7	2	5	—
Diabetes		50	15	35	—
Nephritis		9	5	4	—
Pregnancy diseases of pregnancy or child birth (i.e. Eclampsia)		5	—	5	—
Vascular diseases, including cerebral vascular lesions		163	97	66	—
Intracranial Neoplasm		29	12	17	1
Other diseases of central nervous system		33	17	16	2
Functional disturbances (Hysteria) (malingering)		7	4	3	—
Other general diseases		15	9	6	—
Not classified owing to lack of data		124	73	51	2
	Totals	4078	2015	2063	291

TABLE 4 (APPENDIX E)

SECOND EYE AGE AT ONSET OF EYE TROUBLE

Total	CASES BY AGE GROUPS															
	Under 1		1–4		5–15		16–29		30–39		40–49		50–64		65+	
F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
107	108	94	11	4	7	5	1	3	—	—	—	1	—	—	1	—
76	35	11	21	18	28	22	31	9	11	2	8	5	3	9	—	—
1	1	—	1	1	—	—	—	—	—	—	—	—	1	—	—	—
343	24	42	46	68	118	192	6	9	7	7	8	6	8	14	2	5
1	—	1	—	—	1	—	1	—	—	—	—	—	1	—	—	—
153	1	1	—	1	1	4	6	3	9	5	14	23	62	60	68	56
435	3	3	3	1	5	2	6	2	8	15	25	27	107	153	114	232
21	—	—	1	—	3	1	9	3	4	3	5	5	8	8	3	1
70	70	70	—	—	—	—	—	—	—	—	—	—	—	—	—	—
18	3	1	2	4	4	3	2	3	2	1	1	—	2	4	1	2
82	6	7	7	14	9	11	4	8	8	4	12	13	15	18	10	7
4	1	—	—	1	—	—	—	—	—	—	—	2	—	1	—	—
—	—	—	—	—	1	—	2	—	—	—	—	—	—	—	—	—
128	19	23	12	23	49	56	16	18	3	5	3	2	1	1	—	—
48	1	—	—	—	1	1	6	12	28	11	27	13	35	10	5	1
17	2	—	7	11	5	2	4	1	—	1	1	—	1	2	—	—
8	—	1	3	3	—	3	1	—	—	1	—	—	—	—	—	—
11	1	1	1	—	4	1	2	3	1	5	2	1	2	—	—	—
34	3	1	5	10	9	14	4	3	—	3	—	1	1	2	1	—
2	1	—	1	—	—	—	—	—	—	—	—	1	1	1	—	—
255	2	10	14	16	22	29	33	44	30	35	29	40	38	47	22	34
7	—	—	2	2	—	1	1	3	—	—	2	—	1	1	—	—
2	3	1	—	1	—	—	—	—	—	—	—	—	—	—	—	—
14	1	1	3	1	9	3	3	—	—	2	2	2	3	4	2	1
3	—	—	—	—	3	—	29	—	18	—	15	1	18	1	9	1
—	—	—	—	—	1	—	16	—	7	—	—	—	1	—	—	—
—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	1	—
15	—	—	3	2	11	7	15	1	4	2	2	1	4	2	3	—
—	—	—	—	—	—	—	1	—	—	—	2	—	6	—	2	—
5	—	—	—	—	—	1	—	2	—	—	—	—	1	1	1	1
35	—	—	—	—	1	—	—	—	1	—	2	5	8	24	3	6
4	—	—	—	—	—	—	1	—	—	1	1	2	3	1	—	—
5	—	—	—	—	—	—	—	1	—	3	—	1	—	—	—	—
66	—	—	1	—	2	—	4	1	2	3	6	4	41	28	41	30
17	1	—	1	—	4	1	3	4	—	4	3	8	—	—	—	—
16	2	—	—	1	—	7	3	3	4	2	4	—	3	2	1	1
3	—	1	—	—	—	—	1	—	—	—	1	—	1	2	1	—
6	—	1	—	—	3	1	—	2	—	—	1	—	—	1	5	1
51	2	2	6	6	11	10	10	8	12	4	9	6	16	9	7	6
2063	291	272	152	188	312	377	221	146	159	119	185	170	392	406	303	385
	563		340		689		367		278		355		798		688	

of blindness, the age at onset for the different causes and the age at death. It is suggested that 5 yearly age groups should be adopted, and further subdivision may be found desirable, *e.g.*, to separate infancy, pre-school and school years from the adolescent stage. Further details such as school education, training and occupation, would simplify the application of the Prevention and Treatment of Blindness (Scotland) Act.

TABLE 1. (III and IV)

Age and sex distribution at onset of eye trouble and at onset of blindness.

"Eye Trouble" means the condition which led to blindness. Females are slightly in excess of males, the difference being only 0·58 per cent.

Blindness occurred at 50 years and over in 55·8 per cent., and the age of onset of the condition causing blindness was under that age in 63·5 per cent.

It will be noted that the higher age groups are 50-64 and 65 plus compared with the Glasgow subdivision into 50-59, 60-69, 70 and over.

TABLE I (III & IV)

AGE AND SEX DISTRIBUTION AT ONSET OF EYE TROUBLE ONSET OF BLINDNESS AND PRESENT

Age Group	Number of Persons in Group at					
	Onset Eye Trouble		Onset Blindness		Present Age	
	Male	Female	Male	Female	Male	Female
Under 1	291	272	130	134	—	—
1 – 4	152	188	47	54	17	16
5 – 15	312	377	148	99	57	32
16 – 29	221	146	245	164	179	120
30 – 39	159	119	174	130	152	143
40 – 49	185	170	243	234	255	188
50 – 64	392	406	562	645	649	658
65 + ...	303	385	466	603	706	906
Totals ...	2015	2063	2015	2063	2015	2063

TABLE 2. (VI, VII, B Appendix)

Causes of blindness second eye: at present age.

An excerpt of the more important causes is given below to show the general agreement with the Glasgow figures.

	Rest of Scotland Cases	Per cent.	Glasgow Per cent.
Cataract	706	17·3	16·77
Myopia	563	13·8	16·34
Venereal Disease	525	12·8	13·9
Chronic Septicaemia	445	10·9	10·72
Glaucoma	314	7·6	8·7
Injury	164	4·0	6·4
Others		33·6	27·17
		100·0	100·0

TABLE 3. (Appendix E)

Causes of blindness : second eye : age at onset of blindness.

Sex Incidence.

Table 3 shows the sex incidence for all causes in age groups and the totals are also given.

It is evident that there is little difference in the incidence of blindness between the sexes and the total number of males, 2,015, and females, 2,063, nearly conforms to the proportion of each sex in the population.

Considering the causes separately, there is a significant difference in a number of causes.

The following are commoner in males :— Congenital anomalies and abiotrophies, particularly the latter; acquired syphilis; trauma is very much commoner in men, showing a ratio for all forms of over 5-1; vascular diseases.

The following are commoner in females :— Myopia; cataract; congenital syphilis; septicaemia, diabetes.

Comments on causes of blindness are made under age groups to conform with the Glasgow report.

Pre-School period (0-4 years) :— Congenital anomalies and ophthalmia neonatorum are together chiefly responsible for blindness, accounting for 62·8 per cent. of the total of 366 cases. Congenital syphilis is next in importance with a percentage of 8·4.

School period (5-15 years) :— With a total of 247 cases, this period shows a decidedly lower incidence than the pre-school period.

Congenital syphilis is the commonest cause, with 53 cases accounting for 22·6 per cent. of the total.

Congenital anomalies and abiotrophies show 47 cases=19 per cent.

Sympathetic ophthalmia with 17 cases is the highest incidence of any period.

Blindness is attributed to myopia at this early age in 8 cases, 3·2 per cent. of the total.

Adolescent period (16-29 years):—Total cases 409. The causes in order of frequency are as follows:— Congenital anomalies and abiotrophies 82=20 per cent. Congenital syphilis 54=13·2 per cent. Chronic septicaemia 50=12·2 per cent. Trauma 48=11·7 per cent. Myopia 45=11 per cent. Acquired syphilis makes its first appearance with 10 cases.

Working period (30-49 years):— Total cases 781.

Myopia heads the list of causes with 131 cases, 16·7 per cent. of the total. Chronic septicaemia 118 cases, 15·1 per cent. Abiotrophies 72 cases, 9·2 per cent. Acquired syphilis 63 cases and congenital syphilis 58 cases, 8·06 and 7·4 per cent. respectively. Trauma, including sympathetic ophthalmia, accounts for 56 cases, 7·1 per cent.

50 years and over:— Total cases 2,272. The number of cases in this extensive period shows that 55·7 per cent. of the total cases of blindness occur at 50 years and over.

Cataract takes first place with 663 cases, 29·1 per cent. Myopia 376 cases, 16·3 per cent. Glaucoma, 274, 12 per cent. Chronic septicaemia 240, 10·5 per cent. Vascular diseases 151, 6·6 per cent.

It will be noted that the degenerative diseases are now the prominent causes, cataract showing the highest incidence.

Comparing the figures in this table with the Glasgow report, the greatest difference is in the incidence of acquired syphilis as a cause of blindness. In Glasgow and the South-West acquired syphilis is responsible for 196 cases, or 6·08 per cent. of the total, compared with 151 cases, or 3·7 per cent. in the rest of Scotland.

TABLE 4. (Appendix E)

Causes of blindness: second eye: age at onset of eye trouble.

A table recording the age at onset of eye trouble can profess to a fair degree of accuracy only, as there may be considerable doubt in conditions where the onset is insidious. The history given by the patient is frequently vague or misleading, and the age recorded is the considered opinion of the examiners. Instructions to examiners by the Department of Health for Scotland show an appreciation of the difficulty: "It is expected that examiners will usually be able to obtain the approximate age of onset. The surgeon's opinion is more reliable than the patient's statement."

Myopia is much commonest in the 5-15 period, the age group at which congenital syphilis is also commonest.

Acquired syphilis increases from the 16-29 period with 18 cases to the 50-64 period with 45 cases. There is a sudden rise in the 30-39 period and this corresponds with the Glasgow figures.

Cataract and glaucoma show a sudden increase at 50-64.

Chronic septicaemia is an active cause throughout, with no considerable variation.

The onset in diabetes is almost entirely in the period 50-64, and the vascular diseases show only a few cases until the same period is reached.

TABLE 5. (A Appendix)

Occupations engaged in prior to blindness.

The difficulty in aligning the population at risk with the occupations recorded is discussed under table E for All Scotland, where comments are also made on the occupations in general.

The large difference shown in the figures for spinners, etc., between the rest of Scotland and Glasgow is due to the preponderance of these occupations in the rest of Scotland.

On the other hand, Glasgow employs a much higher proportion of metal workers, and this is reflected in the much higher number of blind persons in this occupation.

TABLE 6. (X and D Appendix)

Average duration from onset to actual blindness of the more important causes.

The figures correspond in general with the Glasgow table X and for most causes the correlation is very close.

The longer average duration in certain cases such as myopia and local infections is accounted for by the estimated earlier age of onset for these conditions in the rest of Scotland.

TABLE 7. (IX)

Causes in relation to duration of blindness.

The duration of disability for different causes brought out in this table is an aspect of great importance both to the blind person and to the State.

Chronic septicaemia, congenital syphilis, congenital anomalies and ophthalmia neonatorum show the highest proportion of "patient years" of blindness.

It it emphasised that this is a proportional table, *e.g.*, ophthalmia neonatorum shows a percentage of 11·3 of the total although the number of cases is comparatively small.

TABLE 5 (APPENDIX A)

OCCUPATIONS ENGAGED IN PRIOR TO BLINDNESS OF THE 4,078 CASES FOR THE REST OF SCOTLAND

	M.	F.
FISHING INDUSTRY	55	—
AGRICULTURAL OCCUPATIONS		
Dairyworkers, Ploughmen	130	17
Others	43	1
MINING AND QUARRYING OCCUPATIONS		
Miners (coal and shale)	205	—
Others	25	1
METAL WORKERS (*not electro-plate or precious metals*)	88	7
ELECTRICAL WORKERS	12	—
TEXTILE WORKERS		
Spinners, Weavers, Winders	81	259
Tailors, Milliners, Tailoresses	37	45
Others	33	9
MAKERS OF FOODS, DRINKS AND TOBACCO		
Bakers	19	1
Others	24	5
WORKERS IN WOOD AND FURNITURE	46	1
BUILDERS, BRICKLAYERS, STONE AND SLATE WORKERS	19	—
BUILDERS' ETC., LABOURERS	39	—
PAINTERS AND DECORATORS, FRENCH POLISHERS	16	—
TRANSPORT AND COMMMUNICATIONS		
Railway Transport Workers	20	—
Road Transport Workers	57	—
Water Transport Workers	30	—
Others	74	1
COMMERCIAL OCCUPATIONS		
Salesmen and Shop Assistants	96	28
Others	32	8
PROFESSIONAL OCCUPATIONS (*excluding Clerical*)	56	29
CLERICAL OCCUPATIONS (*Clerks, Typists, etc.*)	28	12
PERSONS ENGAGED IN PERSONAL SERVICE (*including Institutions, Clubs, Hotels, etc.*)		
Domestic Service	11	198
Others	21	61
OTHER GENERAL OCCUPATIONS	44	43
Labourers	201	2
Housewives	—	952
NO OCCUPATION	473	383
Totals	2015	2063

TABLE 6 (X AND APPENDIX D)

AVERAGE DURATION FROM ONSET TO ACTUAL BLINDNESS OF THE MORE IMPORTANT CASES

Cause of Blindness	Number of Cases	Mean Age Onset	Mean Age Blindness	Average Duration in Years
Myopia	563	12·1	51·6	39·5
Trachoma	35	26·1	50·0	23·9
Phlyctenular Disease	57	15·8	41·5	25·7
Abiotrophy	213	15·5	35·6	20·1
Congenital Anomalies	235	—	14·4	14·4
Chronic Septicaemia	445	37·4	47·6	10·2
Congenital Syphilis	231	11·3	22·1	10·8
Local Infections	158	33·9	49·1	15·2
Ophthalmia Neonatorum	140	1·0	9·9	8·9
Specific Fevers (Smallpox) (Measles) (Scarlet) (Diphtheria)	37	13·3	23·1	9·8
Cataract	706	60·8	65·0	4·2
Diabetes	50	56·8	60·6	3·8
Non Industrial Injury	67	26·2	31·1	4·9
Glaucoma	314	57·8	61·9	4·1
Industrial Injury	95	40·5	42·6	2·1
Sympathetic Ophthalmia	57	25·9	29·4	3·5
Meningitis	12	9·0	11·5	2·5
Vascular Disease	163	60·0	63·8	3·8
Acquired Syphilis	151	43·9	49·3	5·4
Diseases of Central Nervous System	40	33·6	37·2	3·6
Others	309	34·4	50·9	16·5
	4078	33·5	47·1	13·6

TABLE 7 (IX)

CAUSES IN RELATION TO DURATION OF BLINDNESS

Cause of Blindness	Number of Cases	Percentage Distribution	Patient Years of Blindness as Percentage of Total
Others	644	15·8	13·5
Chronic Septicaemia	445	10·9	12·7
Syphilis Congenital	231	5·7	12·1
Congenital Anomalies	235	5·8	11·7
Ophthalmia Neonatorum	140	3·4	11·3
Injury—All Forms	221	5·4	9·0
Myopia	563	13·8	9·0
Cataract	706	17·3	4·6
Local Infections	158	3·9	4·5
Abiotrophy	213	5·2	4·4
Glaucoma	314	7·7	2·8
Syphilis Acquired	151	3·7	2·3
Phlyctenular Disease	57	1·4	2·1
All causes	4078	100·0	100·0

TABLE 8 (XXXII)

CASES WHICH TREATMENT MIGHT REMOVE FROM BLIND CATEGORY

Defect	Number of Cases	Treatment Recommended	Percentage might Improve
Cataract	690	260	37·7
Aphakia	41	11	26·8
Inflammatory Disease of Cornea-Interstitial	147	32	21·8
Optic Neuritis or Atrophy (Primary and Extra-ocular)	349	59	16·9
Cataract (Congenital)	89	14	15·7
Irido-Cyclitis	432	56	13·0
Inflammatory Disease of Cornea-Ulcerative	421	54	12·8
Glaucoma-primary	317	39	12·3
Detachment of Retina	53	3	5·7
Total	2539	528	20·8

TABLE 8. (XXXII)

A selection of the more important conditions where treatment might remove from the blind category.

Out of the total of 4,078 blind persons under review treatment was recommended by the examining surgeons in 703 cases—17·2 per cent. In the great majority of cases the treatment recommended was surgical.

In the Glasgow report it was considered that in 16·2 per cent. of the cases treatment might cure the blindness, the treatment being mainly surgical.

TABLE 9. (L and M Appendix)

Defects in second blind eye with distribution according to sex, age of onset, age of blindness.

This table is given in detail and corresponds with the L and M Glasgow tables.

In the Glasgow report the six defects shown below were responsible for nearly three-quarters of the cases. For the rest of Scotland the same group was responsible for 68·8 per cent. The comparative percentages are given in this excerpt.

The next most important defects found were retinitis degenerative, including pigmentosa 238 cases, 5·84 per cent., and choroiditis, not myopic, 209 cases, 5·13 per cent.

The total congenital defects found was 202 cases of which cataract was commonest with 89 cases, 2·18 per cent.

	Cases	Percentage of Total	Glasgow Percentage
Cataract and Aphakia ...	731	17·93	17·6
Myopia	559	13·71	16·3
Optic Nerve Atrophy ...	349	8·56	11·8
Ulcer of Cornea ...	421	10·32	9·8
Primary Glaucoma ...	317	7·77	8·7
Irido cyclitis	432	10·59	8·7
	2,809	68·88	72·9

TABLE 10. (N Appendix)

Causes of blindness with defects found at examination.

The defects found are shown in their relationship to the different causes of blindness.

Congenital conditions are given in detail and this Table cannot be combined with the Glasgow figures so far as these conditions are concerned.

TABLE 11.

Occurrence of specified disease.

This table is designed to reveal the comparatively high incidence of certain causes of blindness in the different regions.

Cataract as a cause of blindness in the Inverness and Zetland regions is at least partly due to lack of facilities for operative treatment in the sparsely populated country areas.

Myopia shows a significantly high percentage in the Dundee clinic, where women textile workers are commonly affected.

Out of the total of 169 persons blind from myopia 99 were female jute workers, and of these 33 were blind in the age groups 16-49.

Retinitis pigmentosa is unusually common in the Aberdeen area where the subject is at present under investigation. Most cases are sporadic and the heredity of the disease is very complicated. The majority appear to be recessive in character and consanguinity must be a factor even although relationship cannot be traced.

TABLE 9 (APPENDIX L & M)

DEFECTS IN SECOND BLIND EYE WITH DISTRIBUTION ACCORDING TO SEX, AGE OF ONSET OF EYE TROUBLE AND AGE OF BLINDNESS

CONDITION	DISTRIBUTION				AGE OF ONSET OF EYE TROUBLE									AGE AT ONSET OF BLINDNESS								
	Cases	%	Males	%	Birth	1–4	5–15	16–29	30–39	40–49	50–64	65+	N.S.	Birth	1–4	5–15	16–29	30–39	40–49	50–64	65+	N.S.
Wounds of the eyeball	107	2·62	98	4·86	—	3	14	37	18	13	14	8	—	—	—	9	37	19	16	15	11	—
Inflammatory disease of conjunctiva	38	0·93	18	0·89	7	5	8	6	2	1	6	3	—	4	2	5	3	3	3	12	6	—
Inflammatory disease of lids and lacrymal sac	6	0·15	1	0·05	—	1	2	1	—	—	1	1	—	—	—	—	—	—	—	2	4	—
Inflammatory disease of sclera	6	0·15	1	0·05	—	—	2	1	2	—	1	—	—	—	—	—	2	—	2	2	—	—
Inflammatory disease of cornea, ulcerative	421	10·32	202	10·02	146	60	52	27	24	33	52	27	—	86	33	43	30	23	45	96	65	—
Inflammatory disease of cornea, interstitial	147	3·60	72	3·57	16	22	74	26	5	3	1	—	—	5	6	36	32	20	24	20	4	—
Other Corneal disease	22	0·54	6	0·30	7	2	3	4	1	1	4	—	—	2	—	—	3	4	8	4	1	—
Iridocyclitis	432	10·59	189	9·38	22	34	71	81	60	69	67	28	—	5	12	41	68	52	64	138	52	—
Endophthalmitis, Panophthalmitis, Sympathetic	45	1·10	32	1·59	—	5	15	14	4	1	5	1	—	—	3	15	12	6	2	5	2	—
Retinitis, exudative	58	1·42	20	0·99	2	2	3	5	4	7	24	11	—	1	2	1	4	4	5	26	15	—
Retinitis, degenerative inc. Pigmentosa	238	5·84	153	7·59	40	41	45	28	10	16	33	25	—	7	4	20	35	26	42	63	41	—
Detachment of Retina	53	1·30	34	1·69	—	—	3	11	8	11	15	5	—	—	—	3	9	7	10	17	17	—
Choroiditis, not Myopic	209	5·13	96	4·76	25	17	21	25	19	20	45	37	—	15	4	8	27	12	27	61	55	—
Optic neuritis or Atrophy (primary extra-ocular)	349	8·56	232	11·51	36	11	35	53	55	65	63	31	—	26	11	34	44	48	60	86	40	—
Retro-bulbar Neuritis, Toxic Amblyopia	22	0·55	17	0·84	—	—	2	2	5	4	6	3	—	—	—	1	3	3	5	6	4	—
Intermediate	4	0·10	1	0·05	—	2	—	—	—	—	1	1	—	—	2	—	—	—	—	—	2	—
Tumour, intra-ocular	3	0·07	2	—	1	2	—	—	—	—	—	—	—	—	3	—	—	—	—	—	—	—
Cataract	690	16·92	268	13·40	2	4	5	7	22	46	261	343	—	—	—	1	2	7	24	206	450	—
Aphakia	41	1·01	20	0·99	2	—	2	1	4	5	18	9	—	1	—	1	2	3	3	14	17	—
Other lens abnormalities	1	0·02	1	0·05	—	—	—	—	—	—	—	1	—	—	—	—	—	—	—	—	1	—
Myopia	559	13·71	220	10·92	66	115	307	16	14	13	21	7	—	4	4	7	46	43	90	276	89	—
Normal eye and refractive error	13	0·32	6	0·30	1	1	—	1	2	—	6	2	—	—	—	—	2	2	1	5	3	—
Intra-ocular Haemorrhage	29	0·71	17	0·84	—	—	2	5	3	—	9	10	—	—	—	—	5	—	3	7	14	—
Glaucoma, primary	317	7·77	160	7·94	3	2	10	9	12	39	120	122	—	1	—	1	8	10	21	112	164	—
Glaucoma, secondary (Haemorrhagic)	2	0·05	2	0·10	—	—	—	—	—	—	1	1	—	—	—	—	—	—	—	1	1	—
Nystagmus	5	0·12	3	0·15	1	1	1	—	—	—	2	—	—	1	—	1	—	—	—	3	—	—
Cortical and Tract Lesions	15	0·37	6	0·30	2	—	1	2	—	1	6	3	—	1	1	1	1	1	—	5	5	—
Lid Paralysis	1	0·02	—	—	—	—	—	—	1	—	—	—	—	—	—	—	—	1	—	—	—	—
Amblyopia Ex Anopsia	9	0·22	8	0·40	—	1	1	—	—	2	3	2	—	—	—	—	1	—	1	3	4	—
Congenital Anomalies	202	4·93	114	5·66	180	8	9	2	1	2	—	—	—	103	13	19	30	5	19	10	3	—
Others	34	0·83	16	0·79	4	1	1	3	2	3	13	7	—	2	1	—	3	5	2	12	9	—
Total	4078	100	2015	100	563	340	689	367	278	355	798	688	—	264	101	247	409	304	477	1207	1069	—

TABLE 10 (

CAUSES OF BLINDNESS WITH DEFI

CAUSE	Congenital Anomalies	Wounds of the Eyeball	Inflammatory Disease of Conjunctiva	Inflammatory Disease of Lids and Lacrymal Sac	Inflammatory Disease of Sclera	Inflammatory Disease of Cornea Ulcerative	Inflammatory Disease of Cornea Interstitial	Other Corneal Disease	Iridocyclitis
CONGENITAL AND UNDETERMINED									
Congenital anomalies	183	—	—	—	—	1	1	5	2
Abiotrophies, etc. (Retinitis Pigmentosa) (Leber) (Tay Sachs)	4	—	—	—	—	1	—	9	—
Tumour of Globe and Orbit	—	—	—	—	—	—	—	—	—
Myopia	1	—	—	—	—	1	—	—	2
Other Errors of Refraction	—	—	—	—	—	1	—	—	—
Glaucoma, Primary	—	—	—	—	—	—	—	—	—
Cataract, Primary	3	—	—	1	—	—	—	—	1
Other Primary Ocular Defects (Primary Detachment)	—	—	—	—	—	1	—	3	—
INFECTIOUS AND TOXIC									
Ophthalmia Neonatorum	1	—	6	—	—	126	1	1	1
Trachoma	—	—	26	2	—	5	—	2	—
Local Septic infection of Coats of Eye	—	—	—	1	1	141	3	—	4
Other Local Specific infections (Gonorrhoea) (Mycosis)	—	—	—	—	—	3	—	—	—
Gonorrhoea	—	—	—	—	—	3	—	—	2
Syphillis, Congenital	2	—	—	—	—	10	134	—	36
Syphilis, Acquired, including not definitely Congenital	—	—	—	—	—	—	1	—	13
Specific Fevers (Smallpox) (Measles) (Scarlet) (Diphtheria)	—	—	—	—	—	22	1	—	5
Meningitis (Non-Tuberculous) (including Cerebro-Spinal Fever)	—	—	—	—	—	—	—	—	4
Tuberculosis	—	—	—	—	1	4	—	—	17
Phlyctenular, Strumous and similar, not definitely Tuberculous	—	—	—	—	1	50	1	—	3
Septicaemia, Acute	—	—	—	—	—	—	—	—	2
Septicaemia, Chronic, Auto-toxic, focal sepsis	1	—	—	1	3	22	1	—	305
Other General Infectious and Organismal Diseases	—	—	—	—	—	3	—	—	3
TRAUMATIC AND CHEMICAL									
Birth Trauma	1	—	—	—	—	—	—	—	—
Non-industrial Trauma	2	23	—	—	—	6	—	—	1
Industrial Trauma	—	70	2	—	—	8	—	1	3
War Trauma	—	13	—	—	—	6	—	—	—
Trauma, category not ascertainable	—	—	—	—	—	—	—	—	—
Sympathetic Ophthalmia	—	—	—	—	—	2	—	—	14
Chemico-toxic, non-industrial (Tobacco) (Alcohol) (Lead)	—	—	—	—	—	—	—	—	—
Scheduled Industrial Diseases (Lead) (Pyroxylin) (Carbon Bi-sulphide) (Aniline) (Phosphorus) (Glassblowers' Cataract) (Metal Workers' Cataract) (Miners' Nystagmus)	—	—	—	—	—	—	—	—	—
SYSTEMIC DISEASE									
Anaemia and Blood Diseases	—	—	—	—	—	—	—	—	—
Diabetes	—	—	—	1	—	—	—	—	3
Nephritis	—	—	—	—	—	—	—	—	—
Pregnancy, Diseases of Pregnancy or Child Birth, i.e. Eclampsia	—	—	—	—	—	—	—	—	—
Vascular Diseases, including Cerebral Vascular Lesions	—	—	—	—	—	—	—	—	—
Intracranial Neoplasm	1	—	—	—	—	—	—	—	—
Other Diseases of Central Nervous System	—	—	—	—	—	—	—	—	—
Functional Disturbances (Hysteria) (Malingering)	1	—	—	—	—	—	—	—	—
Other General Diseases	—	—	4	—	—	2	—	1	—
Not Classified owing to lack of Data	2	1	—	—	—	4	4	—	11
Total	202	107	38	6	6	421	147	22	432

LE 10 (APPENDIX N)

I DEFECT FOUND AT EXAMINATION DEFECT

Other Corneal Disease	Iridocyclitis	Endophthalmitis, Panophthalmitis, Sympathetic	Retinitis Exudative	Retinitis Degenerative, Including Pigmentosa	Detachment of Retina	Choroiditis, not Myopic	Optic Neuritis or Atrophy (Primary and Extra-ocular)	Retro-bulbar Neuritis Toxic Amblyopia	Indeterminate	Tumour, Extra-ocular	Tumour, Intra-ocular	Cataract	Aphakia	Other Lens Abnormalities	Myopia	Normal Eye and Refractive Error	Intra-ocular Haemorrhage	Glaucoma, Primary	Glaucoma Secondary (Haemorrhagic)	Nystagmus	Cortical and Tract Lesions	Lid Paralysis	Amblyopia Ex Anopsia	Others	Totals
5	2	—	—	6	—	11	20	—	—	—	—	—	2	—	2	—	—	—	—	1	—	—	1	1	235
9	—	1	—	161	1	3	30	3	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	213
—	—	—	—	—	1	—	—	—	—	—	3	—	—	—	—	—	—	—	—	—	—	—	—	—	4
—	2	—	—	—	—	—	2	—	—	—	—	2	—	—	544	—	1	10	—	—	—	—	—	—	563
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	—	—	—	—	—	—	1	—	4
—	—	—	—	—	—	—	—	—	—	—	—	3	—	—	5	—	—	304	1	1	—	—	—	—	314
—	1	—	—	—	—	1	—	—	—	—	—	660	34	—	1	—	—	1	—	1	—	—	—	3	706
3	—	—	—	1	44	—	—	—	—	—	—	—	—	1	—	—	—	—	—	—	—	—	2	2	54
1	1	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	138
2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	35
—	4	—	2	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	153
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	—	—	—	1	5
—	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	5
—	36	—	3	1	—	32	12	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	—	231
—	13	—	2	2	—	26	105	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	1	151
—	5	—	—	—	—	4	3	—	—	—	—	1	—	—	1	—	—	—	—	—	—	—	—	—	37
—	4	—	—	—	—	—	8	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	12
—	17	—	—	—	—	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	24
—	3	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	1	—	—	—	—	—	—	—	57
—	2	—	—	—	—	2	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	5
—	305	1	6	4	3	78	12	3	1	—	—	2	—	—	1	—	—	—	—	—	—	—	—	1	445
—	3	—	—	1	—	1	3	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	13
—	—	—	—	—	—	—	2	—	—	—	—	—	—	—	—	—	—	—	—	—	2	—	—	—	5
—	1	1	—	—	1	—	2	—	—	—	—	—	—	—	1	—	—	—	—	—	—	—	—	—	37
1	3	—	—	—	1	—	4	—	—	—	—	1	2	—	—	1	1	—	—	—	—	—	—	1	95
—	—	—	—	—	—	—	3	—	—	—	—	—	—	—	—	—	1	—	—	—	1	—	—	1	25
—	—	—	—	—	—	—	1	—	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	2
—	14	41	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	57
—	—	—	—	—	—	1	2	4	—	—	—	—	1	—	—	—	—	—	—	—	—	—	3	—	11
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
—	—	—	—	—	—	2	5	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	7
—	3	—	26	3	—	2	1	—	—	—	—	12	1	—	—	—	1	—	—	—	—	—	—	—	50
—	—	—	7	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	9
—	—	—	2	—	—	—	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	5
—	—	—	7	49	—	27	24	2	—	—	—	4	—	—	—	4	23	1	1	—	6	1	—	14	163
—	—	—	—	—	—	—	27	—	—	—	—	—	—	—	—	1	—	—	—	—	—	—	—	—	29
—	—	—	—	—	—	1	24	2	1	—	—	—	—	—	1	—	—	—	—	—	4	—	—	—	33
—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	—	2	1	—	—	—	—	—	—	2	7
1	—	1	—	2	—	3	1	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	15
—	11	—	3	7	2	11	55	6	1	—	—	4	—	—	1	3	—	—	—	1	2	—	2	4	124
2	432	45	58	238	53	209	349	22	4	—	3	690	41	1	559	13	29	317	2	5	15	1	9	34	4078

TABLE II.

Occurrence of Specified Diseases.

Clinic	Total No. of Cases	Cataract	Per cent.	Myopia	Per cent.	Abio-trophies	Per cent.
Aberdeen ...	711	83	11·1	93	13·1	71	10·0
Dumfries ...	96	24	25·0	11	11·5	4	4·2
Dundee ...	928	196	21·1	169	18·2	21	2·3
Edinburgh	2,061	306	14·8	257	12·5	107	5·2
Inverness ...	240	80	30·3	30	12·5	6	2·5

TABLES FOR ALL SCOTLAND.

The tables which follow are the result of amalgamation of the figures for Glasgow and South-West Scotland and the figures published in this report for the rest of Scotland.

The combined tables deal with 7,297 registered blind persons out of an estimated total of between 9,500 and 10,000 registered and unregistered in Scotland.

TABLE A. (ALL SCOTLAND)

Age distribution at onset of eye trouble and at onset of blindness.

The higher age groups are combined as 50 and over, as further subdivision was not possible on account of the different methods of grouping in the two investigations. This applies to all the combined tables.

There is no separate record for males and females for the whole of Scotland but comments are made on sex incidence under Table 3 for the rest of Scotland.

The importance of the first year of life both as regards the onset of conditions causing blindness and the onset of blindness itself is shown in the table, the percentages of the total being 12·7 and 5·8 respectively.

This table emphasises the importance of the lag in registration of blindness. Only one case is registered as blind in the age group O—under 1 although in 420 cases it was stated that blindness occurred under the age of one year.

This question of lag in registration has been discussed in the introduction to this report where it is pointed out that the lag occurs up to the age group 16-29.

TABLE A. (ALL SCOTLAND).

AGE DISTRIBUTION AT ONSET OF EYE TROUBLE. ONSET OF BLINDNESS AND PRESENT

Age Group	Number of Persons in Group at		
	Onset Eye Trouble	Onset Blindness	Present Age
Under 1	924	420	1
1–4	489	205	52
5–15	1,355	452	182
16–29	658	673	530
30–39	541	600	540
40–49	687	912	805
50+...	2,623	4,017	5,187
Not stated	20	18	—
Totals	7,297	7,297	7,297

At the time of examination persons aged 50 years and over accounted for 71·1 per cent. of the cases; ages 16-49 for 25·7 per cent., and ages under 16 for 3·2 per cent.

TABLE B. (ALL SCOTLAND)

Causes of blindness second eye at present age.

The more important causes are given below with the number of cases and percentages.

Cause	Cases	Percentage
Cataract	1246	17·1
Myopia	1089	14·9
Chronic septicæmia	790	10·8
Syphilis congenital and acquired ...	730	10·0
Glaucoma	594	8·1
Others	2848	39·1
	7297	100·0

TABLE B (ALL SCOTLAND).

CAUSES OF BLINDNESS—SECOND EYE. AT PRESENT AGE

Causes	Totals Male and Female	Cases by Age Groups							
		Under 1	1–4	5–15	16–29	30–39	40–49	Over 50	Not Stated
CONGENITAL AND UNDETERMINED									
Congenital Anomalies	388	1	20	64	109	54	44	96	—
Abiotrophies, etc. (Retinitis Pigmentosa) (Leber) (Tay Sachs')	300	—	1	11	39	42	58	149	—
Tumour of Globe and Orbit	8	—	1	2	1	—	1	3	—
Myopia	1,089	—	—	5	56	50	116	862	—
Other Errors of Refraction	5	—	—	—	1	1	—	3	—
Glaucoma, Primary	594	—	—	—	3	7	24	560	—
Cataract, Primary	1,246	—	—	—	5	5	26	1,210	—
Other Primary Ocular Defects (Primary Detachment)	82	—	—	2	5	8	13	54	—
Totals	3,712	1	22	84	219	167	282	2,937	—
INFECTIOUS AND TOXIC									
Exogenous: Ophthalmia Neonatorum	239	—	10	18	43	46	45	77	—
Exogenous: Trachoma	61	—	—	—	3	9	8	41	—
Exogenous: Local Septic Infection of Coats of Eye	234	—	—	2	5	10	18	199	—
Exogenous: Other Local Specific Infections (Gonorrhoea, Mycosis)	8	—	—	—	—	—	2	6	—
Totals	542	—	10	20	51	65	73	323	—
Endogenous: Gonorrhoea	5	—	—	—	—	—	2	3	—
Endogenous: Syphilis, Congenital	383	—	3	20	90	73	72	125	—
Endogenous: Syphilis, Acquired, including not definitely Congenital	347	—	—	—	6	39	87	215	—
Endogenous: Specific Fevers (Smallpox, Measles, Scarlet, Diphtheria)	76	—	1	6	14	10	13	32	—
Endogenous: Meningitis (Non-Tuberculous) including Cerebro-Spinal Fever	51	—	3	10	15	5	7	11	—
Endogenous: Tuberculosis	44	—	—	1	10	9	10	14	—
Endogenous: Phlyctenular, Strumous and Similar, not definitely Tuberculous	141	—	—	5	13	14	18	91	—
Endogenous: Septicaemia, Acute	15	—	—	—	2	3	1	9	—
Endogenous: Septicaemia, Chronic; Auto-toxic, focal sepsis	790	—	3	4	31	52	104	596	—
Endogenous: Other General Infections and Organismal Diseases	44	—	1	1	7	3	6	26	—
Totals	1,896	—	11	47	188	208	320	1,122	—
TRAUMATIC AND CHEMICAL									
Birth Trauma	8	—	3	2	2	1	—	—	—
Non-industrial Trauma	65	—	1	1	9	8	11	35	—
Industrial Trauma	176	—	—	—	10	18	29	119	—
War Trauma	34	—	—	—	1	6	18	9	—
Trauma, Category not ascertainable	17	—	—	—	1	1	1	14	—
Sympathetic Ophthalmia	128	—	—	5	10	16	6	91	—
Chemico-toxic (Non-industrial) (Tobacco, Alcohol, Lead)	18	—	—	—	—	1	—	17	—
Totals	446	—	4	8	33	51	65	285	—
SYSTEMIC DISEASE									
Anaemia and Blood Diseases	8	—	—	—	1	—	1	6	—
Diabetes	93	—	—	—	2	1	4	86	—
Nephritis	14	—	—	—	—	1	1	12	—
Pregnancy, Diseases of Pregnancy or Child Birth (i.e. Eclampsia)	11	—	—	—	1	3	2	5	—
Vascular Diseases including Cerebral Vascular Lesions	263	—	—	—	2	5	4	252	—
Intracranial Neoplasm	65	—	1	10	10	13	11	20	—
Diseases of Central Nervous System	77	—	3	4	8	9	15	38	—
Other General Diseases	23	—	—	3	1	2	5	12	—
Totals	554	—	4	17	25	34	43	431	—
Not Classified Owing to Lack of Data	147	—	1	6	14	15	22	89	—
Totals	7,297	1	52	182	530	540	805	5,187	—

TABLE C (ALL SCOTLA

CAUSES OF BLINDNESS. SECOND EYE. AGE

Causes	Totals Male and Female	Under 1	1–4
CONGENITAL AND UNDETERMINED			
Congenital Anomalies	388	206	22
Abiotrophies, etc. (Retinitis Pigmentosa) (Leber) (Tay Sachs')	300	12	5
Tumour of Globe and Orbit	8	—	5
Myopia	1,089	6	4
Other Errors of Refraction	5	—	—
Glaucoma, Primary	594	—	—
Cataract, Primary	1,246	2	1
Other Primary Ocular Defects (Primary Detachment)	82	—	—
Totals	3,712	226	37
INFECTIOUS AND TOXIC			
Exogenous: Ophthalmia Neonatorum	239	137	44
Exogenous: Trachoma	61	—	1
Exogenous: Local Septic Infection of Coats of Eye	234	3	9
Exogenous: Other Local Specific Infections (Gonorrhoea, Mycosis)	8	—	—
Totals	542	140	54
Endogenous: Gonorrhoea	5	—	—
Endogenous: Syphilis, Congenital	383	22	19
Endogenous: Syphilis, Acquired, Including Not Definitely Congenital	347	—	—
Endogenous: Specific Fevers (Smallpox, Measles, Scarlet, Diphtheria)	76	1	28
Endogenous: Meningitis (Non-Tuberculous) Including Cerebro-Spinal Fever	51	4	17
Endogenous: Tuberculosis	44	1	1
Endogenous: Phlyctenular, Strumous and Similar Not Definitely Tuberculous	141	—	9
Endogenous: Septicaemia, Acute	15	1	2
Endogenous: Septicaemia, Chronic; auto-toxic Focal Sepsis	790	8	12
Endogenous: Other General Infections and Organismal Diseases	44	—	4
Totals	1,896	37	92
TRAUMATIC AND CHEMICAL			
Birth Trauma	8	5	2
Non-industrial Trauma	65	1	1
Industrial Trauma	176	—	—
War Trauma	34	—	—
Trauma, Category Not Ascertainable	17	—	—
Sympathetic Ophthalmia	128	—	3
Chemico-toxic (Non Industrial) (Tobacco, Alcohol, Lead)	18	—	—
Totals	446	6	6
SYSTEMIC DISEASE			
Anaemia and Blood Diseases	8	—	—
Diabetes	93	—	—
Nephritis	14	—	—
Pregnancy, Diseases of Pregnancy or Childbirth (i.e. Eclampsia)	11	—	—
Vascular Diseases Including Cerebral Vascular Lesions	263	—	—
Intracranial Neoplasm	65	1	3
Diseases of Central Nervous System	77	5	3
Other General Diseases	23	—	—
Totals	554	6	6
Not Classified Owing to Lack of Data	147	5	10
Totals	7,297	420	205

L SCOTLAND).

YE. AGE AT ONSET OF BLINDNESS

	Cases by Age Groups						
1	1–4	5–15	16–29	30–39	40–49	Over 50	Not Stated
	22	37	42	14	32	35	—
	5	25	72	42	63	81	—
	5	—	—	—	—	3	—
	4	16	80	83	179	712	9
	—	1	2	—	—	2	—
	—	1	10	22	41	519	1
	1	2	5	14	57	1,163	2
	—	4	15	10	18	35	—
	37	86	226	185	390	2,550	12
	44	15	6	10	12	15	—
	1	3	6	7	8	36	—
	9	18	10	14	34	146	—
	—	1	1	1	2	3	—
	54	37	23	32	56	200	—
	—	2	1	1	—	1	—
	19	107	91	41	55	47	1
	—	—	20	78	98	150	1
	28	15	8	5	3	16	—
	17	18	5	3	4	—	—
	1	6	15	10	3	8	—
	9	24	22	12	25	49	—
	2	3	—	2	3	4	—
	12	42	93	104	126	404	1
	4	7	5	7	3	18	—
	92	224	260	263	320	697	3
	2	1	—	—	—	—	—
	1	16	12	6	7	22	—
	—	1	45	37	43	50	—
	—	1	23	5	3	2	—
	—	2	1	3	3	8	—
	3	36	29	14	14	32	—
	—	—	1	1	2	13	1
	6	57	111	66	72	127	1
	—	1	1	—	1	5	—
	—	—	2	1	5	85	—
	—	—	—	2	3	9	—
	—	—	1	6	2	2	—
	—	—	3	5	11	243	1
	3	9	13	11	17	11	—
	3	13	13	8	12	23	—
	—	6	2	4	2	9	—
	6	29	35	37	53	387	1
	10	19	18	17	21	56	1
	205	452	673	600	912	4,017	18

TABLE D (ALL

CAUSES OF BLINDNESS. SECOND EYE.

	Causes	Totals Male and Female	Under 1
	CONGENITAL AND UNDETERMINED		
	Congenital Anomalies	388	355
	Abiotrophies etc. (Retinitis pigmentosa) (Leber) (Tay Sachs')	300	49
	Tumour of Globe and Orbit	8	1
	Myopia	1,089	130
	Other Errors of Refraction	5	1
	Glaucoma, Primary	594	2
	Cataract, Primary	1,246	8
	Other Primary Ocular Defects (Primary Deteachment)	82	—
	Totals	3,172	546
	INFECTIOUS AND TOXIC		
Exogenous	Ophthalmia Neonatorum	239	239
Exogenous	Trachoma	61	4
Exogenous	Local Septic Infection of Coats of Eye	234	14
Exogenous	Other Local Specific Infections (Gonorrhoea, Mycosis)	8	1
	Totals	542	258
Endogenous	Gonorrhoea	5	—
Endogenous	Syphilis, Congenital	383	51
Endogenous	Syphilis, Acquired, including not definitely Congenital	347	2
Endogenous	Specific Fevers (Smallpox, Measles, Scarlet, Diphtheria)	76	3
Endogenous	Meningitis (Non-Tuberculous) including Cerebro-Spinal Fever	51	7
Endogenous	Tuberculosis	44	2
Endogenous	Phlyctenular, Strumous and Similar, not definitely Tuberculous	141	5
Endogenous	Septicaemia Acute	15	2
Endogenous	Septicaemia Chronic, Auto-toxic Focal Sepsis	790	16
Endogenous	Other General Infections and Organismal Diseases	44	3
	Totals	1,896	91
	TRAUMATIC AND CHEMICAL		
	Birth Trauma	8	7
	Non-Industrial Trauma	65	2
	Industrial Trauma	176	—
	War Trauma	34	—
	Trauma, Category not ascertainable	17	—
	Sympathetic Ophthalmia	128	1
	Chemico-toxic (Non-Industrial) (Tobacco, Alcohol, Lead)	18	—
	Totals	446	10
	SYSTEMIC DISEASES		
	Anaemia and Blood Diseases	8	—
	Diabetes	93	—
	Nephritis	14	—
	Pregnancy, Diseases of Pregnancy or Childbirth (i.e. Eclampsia)	11	—
	Vascular Diseases including Cerebral Vascular Lesions	263	—
	Intracranial Neoplasm	65	2
	Diseases of Central Nervous System	77	5
	Other General Diseases	23	2
	Totals	554	9
	Not Classified Owing to Lack of Data	147	5
	Totals	7,297	919

E D (ALL SCOTLAND).

OND EYE. AGE AT ONSET OF EYE TROUBLE

	Cases by Age Groups							
	Under 1	1–4	5–15	16–29	30–39	40–49	Over 50	Not Stated
	355	15	12	4	—	1	1	—
	49	45	75	75	21	20	14	1
	1	4	—	—	1	—	2	—
	130	152	618	40	32	30	80	7
	1	—	2	1	—	—	1	—
	2	1	6	14	24	78	468	1
	8	4	12	18	47	96	1,059	2
	—	1	5	20	12	18	26	—
	546	222	730	172	137	243	1,651	11
	239	—	—	—	—	—	—	—
	4	7	19	9	7	1	14	—
	14	26	35	17	21	35	86	—
	1	1	1	1	—	3	1	—
	258	34	55	27	28	39	101	—
	—	—	2	3	—	—	—	—
	51	49	197	58	13	11	4	—
	2	—	3	38	104	99	100	1
	3	36	25	5	2	1	4	—
	7	18	16	5	2	2	—	1
	2	4	16	9	8	3	2	—
	5	41	71	13	4	3	4	—
	2	3	2	—	1	5	2	—
	16	38	102	141	115	121	254	3
	3	12	5	8	3	6	6	1
	91	201	439	280	252	251	376	6
	7	1	—	—	—	—	—	—
	2	7	22	5	3	8	18	—
	—	—	3	53	39	39	42	—
	—	—	1	23	8	1	1	—
	—	2	1	2	4	2	6	—
	1	5	40	30	12	16	24	—
	—	—	—	3	—	2	12	1
	10	15	67	116	66	68	103	1
	—	—	1	2	—	—	5	—
	—	—	1	2	1	11	78	—
	—	—	—	1	2	4	7	—
	—	—	—	1	8	2	—	—
	—	1	2	5	8	21	225	1
	2	2	10	16	8	20	7	—
	5	4	15	13	10	11	19	—
	2	—	6	4	2	1	8	—
	9	7	35	44	39	70	349	1
	5	13	30	19	19	16	44	1
	919	492	1,356	658	541	687	2,624	20

These five specified causes together account for 60·3 per cent. of blindness.

TABLE C. (ALL SCOTLAND)

Causes of blindness second eye. Age at onset of blindness.

AGE GROUPS.

0-4 years :—625 cases.

Congenital anomalies 228 cases, and ophthalmia neonatorum 181 cases, are chiefly responsible for blindness, accounting for 65·4 per cent. of the total.

Congenital syphilis is next in importance with 6·5 per cent.

5-15 years :—452 cases.

Apart from the age group 1-4 this group shows the lowest incidence of any period.

Congenital syphilis is the commonest cause with 107 cases, 23·7 per cent. of the total.

Congenital anomalies show 62 cases, 13·7 per cent.

Sympathetic ophthalmia with 36 cases shows the highest incidence of any age group.

Myopia is the cause at this early age in 16 cases.

16-29 years :—673 cases.	cases	per cent.
Congenital anomalies	114	16·9
Trauma including sympathetic ophthalmia	110	16·3
Chronic septicaemia	93	13·8
Congenital syphilis	91	13·5
Myopia	80	11·9

Acquired syphilis makes its first appearance with 20 cases.

30-39 years :—600 cases.		
Chronic septicaemia	104	17·3
Myopia	83	13·8
Syphilis acquired	78	13·0
Trauma	65	10·8
Congenital anomalies	56	9·3
Syphilis congenital	41	6·8

40-49 years :—912 cases.		
Myopia	179	19·6
Chronic septicaemia	126	13·8
Syphilis acquired	98	10·7
Syphilis congenital	55	6·0
Congenital anomalies	95	10·4
Trauma all forms	70	7·7
Cataract	57	6·2
Glaucoma	41	4·5

50 and over :—4,014 cases.

Cataract	1,163	28·9
Myopia	712	17·7
Glaucoma	519	12·9
Chronic septicaemia	404	10·0
Vascular diseases	243	6·0

Analysis of the age distribution of the different causes shows that congenital anomalies and ophthalmia neonatorum account for blindness in 65·4 per cent. of the cases occurring in the first five years of life.

Syphilis, congenital and acquired, is a major cause from 16 to 49 years and is the commonest cause in the age group 30-39 with 19·8 per cent.

As would be expected, the degenerative diseases are the commonest causes of blindness with advancing years, and cataract, myopia and glaucoma together account for 59·6 per cent. of cases onsetting at 50 years and over.

TABLE D. (ALL SCOTLAND)

Causes of blindness second eye. Age at onset of eye trouble.

Under Table 4, the corresponding table for the rest of Scotland, it is pointed out that only a fair degree of accuracy is possible in the data here presented as there is often considerable doubt in the history given by the patient.

In a large number of cases, 919, the onset of eye trouble is attributed to the first year of life, the outstanding causes being congenital anomalies and ophthalmia neonatorum.

There is a decided drop in numbers in the age group 1-4 with myopia as the principal cause.

The onset of myopia is most frequent at 5-15 years, and congenital syphilis is commoner in this age group than in any other.

There is a sudden increase in acquired syphilis at 30-39 and this is maintained in the 40-49 group.

Chronic septicaemia is common from 5-15 throughout life with little variation.

Cataract, glaucoma, vascular diseases and diabetes are uncommon until the age of 50 years.

TABLE E. (ALL SCOTLAND)

Occupations engaged in prior to blindness.

This table is a statement of the facts given in the certificates of blindness.

Difficulty always exists in aligning the population at risk with the occupations recorded in an investigation such as this which covers practically the whole population.

TABLE E (ALL SCOTLAND).

Occupations engaged in prior to Blindness.

	M	F
FISHING INDUSTRY	61	—
AGRICULTURAL OCCUPATIONS		
Dairyworkers, Ploughmen	152	28
Others	63	1
MINING AND QUARRYING OCCUPATIONS		
Miners (coal and shale)	401	1
Others	62	5
METAL WORKERS (NOT ELECTRO PLATE OR PERVIOUS METALS)	387	7
Electrical Workers	15	—
Textile Workers		
Spinners, Weavers, Winders	85	270
Tailors, Milliners, Tailoresses	71	72
Others	80	62
Makers of Foods, Drinks and Tobacco	30	2
Bakers	30	2
Others	34	7
Workers in Wood and Furniture	105	3
Builders, Bricklayers, Stone and Slate Workers	49	—
Builders' etc., labourers	43	—
Painters and Decorators, French Polishers	29	6
TRANSPORT AND COMMUNICATIONS		
Railway Transport Workers	64	1
Road Transport Workers	117	—
Water Transport Workers	83	1
Others	79	3
COMMERCIAL OCCUPATIONS		
Salesmen and Shop Assistants	152	49
Others	226	25
PROFESSIONAL OCCUPATIONS (EXCLUDING CLERICAL)	96	54
Clerical Occupations (Clerks, Typists, etc.)	55	17
PERSONS ENGAGED IN PERSONAL SERVICE (INCLUDING INSTITUTIONS, CLUBS, HOTELS, ETC.)		
Domestic Service	11	243
Others	37	107
OTHER GENERAL OCCUPATIONS	44	43
Labourers	373	2
Housewives	—	1,953
NO OCCUPATION	743	582
Totals	3,747	3,550

For information on the numbers at risk in each occupation the source is the 1931 census. In the census, subdivision by occupation and industry results in a fairly precise classification of the population; here, however, a statement has been obtained on occupation

only, and for that reason no attempt has been made to calculate the ratio to the appropriate population at risk.

In the table a large number, 856, is returned as of no occupation. This group comprises children, persons who became blind before entering an occupation, and persons who had been blind for a long time and regarded themselves as no longer employable.

In women, the highest number of blind occurs in housewives. This number undoubtedly covers all the older women whether married or single, who have ceased to be engaged in any other occupation.

For men with specified occupations the greatest number occurs in mining with 401, followed by metal workers 387, and labourers 373.

For women apart from housewives the main occupations are spinners, weavers and winders with 276, and domestic service with 243.

The figures suggest a higher rate of blindness in miners and metal workers than in other occupations in the aggregate in Scotland.

The proportion of spinners, weavers and winders shown blind is much higher than the proportion of these occupations in the female population.

While no precise application of the census figures can be made to determine incidence rates, the figures are suggestive that these three occupations (miners, metal workers and spinners, etc.) have a higher than average incidence of blindness.

Table E also shows the relative sex distribution of the registered blind. In Scotland the population comprises slightly more females than males while the blind population shows the reverse. The consequential higher incidence of blindness in the male sex may be largely accounted for by the greater exposure to injury as the cause of blindness. In Table 2 (*i.e.*, for Scotland other than Glasgow and the South-West) traumatic and chemical conditions show 232 cases of which 198 are for males.

TABLE F. (ALL SCOTLAND)

Average duration from onset to actual blindness of the more important causes.

The average duration in years shows for the different causes the length of the twilight period with progressive deterioration in sight until actual blindness supervenes.

Myopia, trachoma and phlyctenular disease head the list with the longest periods of increasing disability, and industrial injury comes last with an average duration of 2·4 years.

Cataract, with the largest number of cases, shows an average duration of 4·6 years.

The table illustrates that the time available for treatment for a number of causes, *e.g.*, glaucoma, is short.

TABLE F (ALL SCOTLAND).

Average Duration from Onset to Actual Blindness of the more Important Causes.

Cause of Blindness	Number of Cases	Mean Age Onset	Mean Age Blindness	Average Duration in Years
Myopia	1,089	14·1	52·1	37·9
Phlyctenular	141	12·3	37·6	25·3
Trachoma	61	25·4	50·0	24·6
Abiotrophy	300	17·0	36·7	19·7
Congenital Anomalies	388	0·4	14·4	14·0
Local Infections	234	36·0	49·5	13·5
Chronic Septicaemia	790	35·9	47·0	11·2
Congenital Syphilis	383	12·4	23·1	10·7
Ophthalmia Neonatorum	237	1·0	9·5	8·5
Specific Fevers (Smallpox, Measles, Scarlet, Diphtheria)	65	10·1	18·4	8·3
Cataract	1,246	60·7	65·3	4·6
Non-industrial Injury	95	27·8	32·4	4·6
Diabetes	93	56·7	60·8	4·2
Glaucoma	594	58·3	61·9	3·5
Vascular Disease	263	60·3	63·6	3·3
Acquired Syphilis	347	42·9	46·2	3·3
Sympathetic Ophthalmia	128	29·4	32·5	3·1
Diseases of Central Nervous System	77	31·0	33·7	2·7
Meningitis	51	10·0	12·6	2·6
Industrial Injury	176	39·3	41·7	2·4

TABLE G. (ALL SCOTLAND)

Causes in relation to duration of blindness.

The duration of blindness for the different causes is an important aspect of the subject both from the social and personal points of view.

The relative importance of most causes of blindness is entirely changed when the duration in years is considered, and it will be seen from the table that the percentage distribution bears no relation to the "patient years" of blindness.

This is well illustrated in the case of cataract, the cause of the greatest number of cases, which is relegated to seventh place with 5·0 years of blindness.

A new significance is given to causes acting in the early years of life and chronic septicaemia, congenital anomalies, ophthalmia neonatorum and congenital syphilis become the prominent group, together accounting for 54·5 per cent. of the total years of blindness. Myopia, which also affects the early years, takes an intermediate place with 9·5 years.

TABLE G (ALL SCOTLAND).

Causes in Relation to Duration of Blindness.

Cause of Blindness.	Number of Cases	Percentage Distribution	Patient Years of Blindness as Percentage of Total
Chronic Septicaemia	790	10·8	12·3
Congenital Anomalies	388	5·3	11·5
Ophthalmia Neonatorum	237	3·2	10·7
Injury—All Forms	428	5·9	10·0
Syphilis Congenital	383	5·3	10·0
Myopia	1,089	14·9	9·5
Cataract	1,246	17·1	5·0
Local Infections	239	3·3	4·0
Abiotrophy	300	4·1	3·7
Phlyctenular Disease	141	1·9	3·4
Glaucoma	594	8·1	3·1
Syphilis Acquired	347	4·8	3·0

TABLE H. (ALL SCOTLAND)

Causes of blindness with defects found at examination.

The defects found are given in their relation to the different causes of blindness.

Numerically the two most important defects were cataract and aphakia with 1,299 cases and myopia with 1,084 cases; 17·8 and 14·9 per cent. respectively. Other defects in order of frequency were as follows:—

	cases	per cent.
Ulceration of the cornea	737	10·0
Optic neuritis and atrophy	729	10·0
Iridocyclitis	711	9·7
Glaucoma primary	597	8·2

Ulceration of the cornea. The commonest causes were:—

Local septic infection	216	29·1
Ophthalmia neonatorum	190	25·7
Phlyctenular disease	133	18·0

Interstitial keratitis caused blindness in 242 cases, 3·3 per cent., the great majority (215) being due to congenital syphilis.

Optic nerve atrophy. Acquired syphilis was the main cause with 236 cases, 32·3 per cent. Other causes were as follows:—

	cases	per cent.
Intracranial neoplasm	61	8·3
Other diseases of the C.N.S.	57	7·8
Vascular disease	52	7·1
Abiotrophies	48	6·5
Chronic septicaemia	42	5·7
Specific fevers	40	5·4

TABLE H (ALL SCOTLAND). *Causes of Blindness with Defects found at Examination*

Cause of Blindness	Congenital Anomalies	Wounds of Eyeball	Inflammatory Diseases of Conjunctiva	Inflammatory Diseases of Lids and Lacrymal Sac	Inflammatory Disease of Sclera	Inflammatory Disease of Cornea Ulcerative	Inflammatory Disease of Cornea Interstitial	Other Corneal Disease	Iridocyclitis	Endophthalmitis	Retinitis, Exudative	Retinitis, Degenerative	Detachment of Retina	Choroiditis (not Myopic)	Optic Neuritis or Atrophy	Retrobulbar Neuritis	Indeterminate	Tumour, Extraocular	Tumour, Intraocular	Cataract
A. Congenital and Undetermined Conditions																				
Congenital Anomalies	330	—	—	—	—	—	1	5	2	—	—	6	—	11	25	—	—	—	—	—
Abiotrophies	4	—	—	—	—	1	—	11	—	1	—	227	1	4	48	3	—	—	—	—
Tumour of Globe and Orbit	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	2	5	—
Myopia	1	—	—	—	—	1	—	—	2	—	—	—	—	—	2	—	—	—	—	2
Other Errors of Refraction	—	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Glaucoma, Primary	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	3
Cataract, Primary	3	—	—	1	—	—	—	—	1	—	—	—	—	1	—	—	—	—	—	1,126
Other Primary Defects	—	—	—	—	—	1	—	3	—	—	—	1	72	—	—	—	—	—	—	—
	338	—	—	1	—	4	1	19	5	1	—	234	74	16	75	3	—	2	5	1,131
B. Infectious and Toxic																				
Ophthalmia Neonatorum	1	—	43	—	—	190	1	1	1	—	—	—	—	1	—	—	—	—	—	—
Trachoma	—	—	40	4	—	15	—	2	—	—	—	—	—	—	—	—	—	—	—	—
Local Septic Infection of Coats of Eye	—	—	2	5	1	216	3	—	4	—	2	—	—	1	—	—	—	—	—	—
Other Local Specific Infections	—	—	1	—	—	5	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	1	—	86	9	1	426	4	3	5	—	2	—	—	2	—	—	—	—	—	—
Gonorrhoea	—	—	1	—	—	2	—	—	2	—	—	—	—	—	—	—	—	—	—	—
Syphilis, Congenital	2	—	—	—	—	10	215	—	59	—	3	9	—	58	25	—	1	—	—	—
Syphilis, Acquired	—	—	—	—	—	—	6	—	35	—	2	12	—	54	236	—	—	—	—	1
Specific Fevers (Smallpox, Measles)	—	—	—	2	—	48	1	—	8	5	—	—	—	5	4	—	—	—	—	2
Meningitis (Non-Tuberculous)	—	—	—	—	—	—	—	—	6	3	—	—	—	—	40	—	—	—	—	—
Tuberculosis	—	—	—	—	2	13	2	—	22	—	—	—	—	2	3	—	—	—	—	—
Phlyctenular, Strumous and Similar	—	—	—	—	1	133	1	1	3	—	—	—	—	—	—	—	—	—	—	—
Septicaemia, Acute	—	—	—	—	—	3	—	—	4	5	—	—	—	2	1	—	—	—	—	—
Septicaemia, Chronic, auto-toxic	1	—	—	1	3	40	7	6	516	2	11	23	4	124	42	3	1	—	—	2
Other General Infections and Organismal diseases	—	—	—	—	—	5	1	2	3	—	2	4	—	3	18	1	2	—	—	—
	3	—	1	3	6	254	233	9	658	15	18	48	4	248	369	4	4	—	—	5
Traumatic and Chemical																				
Birth Trauma	1	—	—	—	—	—	—	—	—	—	—	—	—	—	3	—	—	—	—	—
Non-industrial Trauma	2	37	—	—	—	12	—	—	1	1	—	—	2	—	7	—	—	—	—	—
Industrial Trauma	—	126	2	—	—	21	—	1	3	—	—	—	5	—	11	—	1	—	—	1
War Trauma	—	20	—	—	—	6	—	—	1	—	—	—	—	—	4	—	—	—	—	—
Trauma, not ascertainable	—	9	—	—	—	3	—	—	—	—	—	—	—	—	3	—	—	—	—	—
Sympathetic Ophthalmia	—	—	—	—	—	2	—	—	20	106	—	—	—	—	—	4	—	—	—	—
Chemico-toxic, Non-industrial	—	—	—	—	—	—	—	—	1	—	—	1	—	1	2	5	—	—	—	—
	3	192	2	—	—	44	—	1	26	107	—	1	7	1	30	9	1	—	—	1
Systemic Disease																				
Anaemia and Blood Diseases	—	—	—	—	—	—	—	—	—	—	—	—	—	2	6	—	—	—	—	—
Diabetes	—	—	—	1	—	—	—	—	3	—	40	3	—	2	3	—	—	—	—	36
Nephritis	—	—	—	—	—	—	—	—	—	—	11	1	—	—	—	—	—	—	—	—
Diseases of Pregnancy	—	—	—	—	—	—	—	—	1	2	3	—	—	—	4	—	—	—	—	—
Vascular Diseases	—	—	—	—	—	—	—	—	—	—	29	73	—	32	52	3	—	—	—	4
Intracranial Neoplasm	1	—	—	—	—	—	—	—	—	—	—	—	—	—	61	—	—	1	—	—
Other Diseases of Central Nervous System	—	—	—	—	—	—	—	—	—	—	—	—	—	1	57	3	2	—	—	—
Functional Disturbances and Other General Diseases	1	—	4	—	—	5	—	1	—	1	—	2	—	3	5	1	—	—	—	—
Not classified owing to lack of Data	2	1	—	—	—	4	4	—	13	—	4	8	3	11	67	6	6	—	—	4
	4	1	4	1	—	9	4	1	17	3	87	87	3	51	255	13	8	1	—	44
All Cases	349	193	93	14	7	737	242	33	711	126	107	370	88	318	729	29	13	3	5	1,181

TABLE H (ALL SCOTLAND). *Causes of Blindness with Defects found at Examinatio*

Inflammatory Diseases of Conjunctiva	Inflammatory Diseases of Lids and Lacrymal Sac	Inflammatory Disease of Sclera	Inflammatory Disease of Cornea Ulcerative	Inflammatory Disease of Cornea Interstitial	Other Corneal Disease	Iridocyclitis	Endophthalmitis	Retinitis, Exudative	Retinitis, Degenerative	Detachment of Retina	Choroiditis (not Myopic)	Optic Neuritis or Atrophy	Retrobulbar Neuritis	Indeterminate	Tumour, Extraocular	
—	—	—	—	1	5	2	—	—	6	—	11	25	—	—	—	
—	—	—	1	—	11	—	1	—	227	1	4	48	3	—	—	
—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	2	
—	—	—	1	—	—	2	—	—	—	—	—	2	—	—	—	
—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
—	1	—	—	—	—	1	—	—	—	—	1	—	—	—	—	
—	—	—	1	—	3	—	—	—	1	72	—	—	—	—	—	
—	1	—	4	1	19	5	1	—	234	74	16	75	3	—	2	
43	—	—	190	1	1	1	—	—	—	—	1	—	—	—	—	
40	4	—	15	—	2	—	—	—	—	—	—	—	—	—	—	
2	5	1	216	3	—	4	—	2	—	—	1	—	—	—	—	
1	—	—	5	—	—	—	—	—	—	—	—	—	—	—	—	
86	9	1	426	4	3	5	—	2	—	—	2	—	—	—	—	
1	—	—	2	—	—	2	—	—	—	—	—	—	—	—	—	
—	—	—	10	215	—	59	—	3	9	—	58	25	—	1	—	
—	—	—	—	6	—	35	—	2	12	—	54	236	—	—	—	
—	2	—	48	1	—	8	5	—	—	—	5	4	—	—	—	
—	—	—	—	—	—	6	3	—	—	—	—	40	—	—	—	
—	—	2	13	2	—	22	—	—	—	—	2	3	—	—	—	
—	—	1	133	1	1	3	—	—	—	—	—	—	—	—	—	
—	—	—	3	—	—	4	5	—	—	—	2	1	—	—	—	
—	1	3	40	7	6	516	2	11	23	4	124	42	3	1	—	
—	—	—	5	1	2	3	—	2	4	—	3	18	1	2	—	
1	3	6	254	233	9	658	15	18	48	4	248	369	4	4	—	
—	—	—	—	—	—	—	—	—	—	—	—	3	—	—	—	
—	—	—	12	—	—	1	1	—	—	2	—	7	—	—	—	
2	—	—	21	—	1	3	—	—	—	5	—	11	—	1	—	
—	—	—	6	—	—	1	—	—	—	—	—	4	—	—	—	
—	—	—	3	—	—	—	—	—	—	—	—	3	—	—	—	
—	—	—	2	—	—	20	106	—	—	—	—	—	4	—	—	
—	—	—	—	—	—	1	—	—	1	—	1	2	5	—	—	
2	—	—	44	—	1	28	107	—	1	7	1	30	9	1	—	
—	—	—	—	—	—	—	—	—	—	—	2	6	—	—	—	
—	1	—	—	—	—	3	—	40	3	—	2	3	—	—	—	
—	—	—	—	—	—	—	—	11	1	—	—	—	—	—	—	
—	—	—	—	—	—	1	2	3	—	—	—	4	—	—	—	
—	—	—	—	—	—	—	—	29	73	—	32	52	3	—	—	
—	—	—	—	—	—	—	—	—	—	—	—	61	—	—	1	
—	—	—	—	—	—	—	—	—	—	—	1	57	3	2	—	
4	—	—	5	—	1	—	1	—	2	—	3	5	1	—	—	
—	—	—	4	4	—	13	—	4	8	3	11	67	6	6	—	
4	1	—	9	4	1	17	3	87	87	3	51	255	13	8	1	
93	14	7	737	242	33	711	126	107	370	88	318	729	29	13	3	

Indeterminate	Tumour, Extraocular	Tumour, Intraocular	Cataract	Aphakia	Other lens Abnormalities	Myopia	Intraocular Haemorrhage	Glaucoma, Primary	Glaucoma, Secondary	Nystagmus	Cortical and Tract Lesions	Lid Paralysis	Amblyopia Ex Anopsia	Others	Totals
—	—	—	—	2	—	2	—	—	—	2	—	—	1	1	388
—	2	5	—	—	—	—	—	—	—	—	—	—	—	—	300
—	—	—	2	—	—	1,069	—	—	—	—	—	—	—	—	8
—	—	—	3	—	—	5	1	10	—	—	—	—	2	2	1,089
—	—	—	1,126	108	—	1	—	584	1	1	—	—	—	3	5
—	—	—	—	—	1	—	—	1	—	1	—	—	2	2	594
—	2	5	1,131	111	1	1,077	1	595	1	4	—	—	5	9	1,246
															82
—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	239
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	61
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	234
—	—	—	—	—	—	1	—	—	—	—	—	—	—	1	8
—	—	—	—	—	—	1	—	—	—	1	—	—	—	1	542
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	5
1	—	—	—	—	—	—	—	1	—	—	—	—	—	—	383
—	—	—	1	—	—	—	—	—	—	—	—	—	—	1	347
—	—	—	2	—	—	1	—	—	—	—	—	—	—	—	76
—	—	—	—	—	—	—	—	—	—	—	1	1	—	—	51
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	44
—	—	—	—	—	—	1	1	—	—	—	—	—	—	—	141
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	15
1	—	—	2	—	—	1	2	—	—	—	—	—	—	1	790
2	—	—	—	—	—	—	—	—	—	—	—	—	—	3	44
4	—	—	5	—	—	3	3	1	—	—	1	1	—	5	1,896
—	—	—	—	—	—	—	—	—	—	—	4	—	—	—	8
—	—	—	—	—	1	1	—	—	—	—	—	—	—	1	65
1	—	—	1	2	—	—	1	—	—	—	—	—	—	2	176
—	—	—	—	—	—	—	1	—	—	—	1	—	—	1	34
—	—	—	—	1	—	—	—	—	—	—	1	—	—	—	17
—	—	—	—	—	—	—	—	—	—	—	—	3	—	—	132
—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	14
1	—	—	1	4	1	1	2	—	—	—	6	3	—	4	446
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	3
—	—	—	36	3	—	—	1	—	—	—	1	—	—	—	93
—	—	—	—	—	—	—	—	—	—	—	1	—	—	1	14
—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	11
—	—	—	4	—	—	—	25	1	4	—	19	1	—	20	263
1	—	—	—	—	—	—	—	—	—	—	1	—	—	1	65
2	—	—	—	—	—	1	1	—	—	—	6	1	—	4	77
—	—	—	—	—	—	—	1	—	—	—	—	—	—	—	23
3	—	—	4	—	—	1	—	—	—	1	2	—	2	8	147
3	1	—	44	3	—	2	28	1	4	1	30	2	2	35	701
3	3	5	1,181	118	2	1,084	34	597	5	6	37	3	10	53	7,297

	cases	per cent.
Congenital anomalies	25	3·4
Congenital syphilis	25	3·4

Iridocyclitis. Chronic septicaemia was the main cause with 516 cases, 72·6 per cent. Other causes were :—

Congenital syphilis	59	8·3
Acquired syphilis	35	4·9
Tuberculosis	22	3·1
Sympathetic ophthalmia	20	2·8

TABLE K (ALL SCOTLAND).

DEFECTS IN SECOND BLIND EYE ACCORDING TO AGE OF BLINDNESS

CONDITION	CASES	AGE AT ONSET OF BLINDNESS							
		Under 1	1–4	5–15	16–29	30–39	40–49	50+	Not Stated
Congenital Anomalies	349	186	23	31	37	10	30	32	—
Wounds of Eyeball	193	—	—	11	59	42	35	46	—
Inflammatory Disease of Conjunctiva	93	19	18	8	6	7	4	31	—
Inflammatory Disease of Lids and Lacrymal Sac	14	—	—	—	—	—	2	12	—
Inflammatory Disease of Sclera	7	—	—	—	2	1	2	2	—
Inflammatory Disease of Cornea, Ulcerative	737	121	70	80	59	53	84	270	—
Inflammatory Disease of Cornea, Interstital ...	242	6	10	68	57	32	36	33	—
Other Corneal Disease	33	2	—	—	3	4	10	14	—
Iridocyclitis	711	8	17	62	107	98	110	306	3
Endophthalmitis, etc.	126	2	10	33	26	13	16	26	—
Retinitis, Exudative	107	1	2	1	6	8	9	80	—
Retinitis, Degenerative	370	9	6	26	54	41	74	159	1
Detachment of Retina	88	—	—	4	17	11	19	37	—
Choroiditis	318	16	5	18	36	34	45	164	—
Optic Neuritis or Atrophy	729	38	27	83	87	109	140	244	1
Retro-bulbar Neuritis	29	—	—	2	4	3	5	15	—
Indeterminate	13	—	4	2	1	—	2	3	1
Tumour, Extra-Ocular	3	—	—	—	—	—	—	3	—
Tumour, Intra-Ocular	5	—	5	—	—	—	—	—	—
Cataract	1,181	—	1	1	3	12	46	1,117	1
Aphakia	118	1	—	1	3	5	12	95	1
Other Lens Abnormalities	2	—	—	—	—	1	—	1	—
Myopia	1,084	4	4	15	81	87	177	707	9
Intra-ocular Haemorrhage	34	—	—	1	6	1	4	22	—
Glaucoma	597	1	—	1	10	20	43	521	1
Glaucoma (Secondary)	5	—	—	—	—	—	1	4	—
Nystagmus	6	1	—	1	—	—	1	3	—
Cortical and Tract Lesions	37	3	2	2	2	1	—	27	—
Lid Paralysis	3	—	—	—	—	1	1	1	—
Amblyopia ex Anopsia	10	—	—	1	1	—	1	7	—
Others	53	2	1	—	5	7	3	35	—
All Cases	7,297	420	205	452	673	600	912	4,017	18

TABLE K. (ALL SCOTLAND)

Defects in second blind eye according to age group of onset of blindness.

Cataract and *Glaucoma* are defects of age and the onset of blindness resulting therefrom occurs at the age of 50 and over in the great majority of cases.

Myopia. Blindness is delayed until the age of 50 in 65 per cent. of the cases.

Ulceration of the Cornea. Blindness arises mainly at the two extremes of age.

Interstitial Keratitis. Blindness is spread over from the age group 5-15 and upwards.

Irido-cyclitis. The age of blindness is evenly distributed in the three age groups 16-49.

Optic Nerve Atrophy. The onset of blindness is initially heavy in the age group under 1, reaches the lowest incidence in the age group 1-4, thereafter increasing gradually to the highest incidence in the age group 50 and over.

Wounds of the Eyeball. The highest incidence occurs between the ages of 16 and 39 years.

SUMMARY.

A statistical analysis has been made of 4,087 persons certified blind at the regional clinics for Edinburgh, Dundee, Aberdeen and Inverness, and the results have been compared with the analysis for Glasgow and South-West Scotland published by Marshall and Seiler in 1942[1].

In general there is close agreement between the findings of the two investigations, and tables have been amalgamated to present as complete a record as possible of the blind position in the whole of Scotland.

The combined figures deal with 7,297 certified blind persons, but it has been estimated that the total number of the blind in Scotland is between 9,500 and 10,000.

It has been shown that the chief causes of blindness were cataract, 17·1 per cent.; myopia, 14·9 per cent.; chronic septicaemia, 10·8 per cent.; syphilis, 10·0 per cent.; and glaucoma, 8·1 per cent. Injury was responsible for only 5·9 per cent. of the cases.

An important consideration is the duration of blindness for each cause, as the percentage distribution bears no relation to the "patient years" of blindness. A new significance is given to causes operating in the early years of life and the most important causes from this point of view are chronic septicaemia, congenital anomalies, ophthalmia neonatorum and congenital syphilis which

together account for 44·5 per cent. of the total duration of blindness. Cataract, the cause of the greatest number of cases, is relegated to seventh place, with an average of 5·0 years of blindness.

Of the persons blind, 71 per cent. were over 50 years of age. Table B (All Scotland) shows 3·3 per cent. as blind in the age groups 0-15 but this is a considerable underestimation due to the lag in registration under the age of 16.

The eye trouble which led to blindness had its onset before the age of 50 in 64 per cent. of cases.

Analysis of the age distribution of the different causes shows that congenital anomalies and ophthalmia neonatorum cause blindness in 65·4 per cent. of cases in the first four years of life. Congenital syphilis is the commonest cause in the age group 5-15, accounting for 23·7 per cent. Next in importance is congenital anomalies with 13·7 per cent.

In the age group 16-29 injuries are prominent with 16·3 per cent. The other main causes in this group are congenital anomalies 16·9 per cent., chronic septicaemia 13·8 per cent., congenital syphilis 13·5 per cent., and myopia 11·9 per cent.

In the age group 30-39 chronic septicaemia takes first place with 17·3 per cent. and myopia is the cause in 13·8 per cent. Acquired syphilis 13·0 per cent. is the highest incidence in any group. Congenital anomalies drop to 9·3 per cent., and congenital syphilis with 6·8 per cent. is only half the percentage in the 16-29 age group.

Myopia is an important cause from age 40 onwards, and cataract, myopia and glaucoma together cause blindness in 59·6 per cent. at 50 years and over.

Occupations are discussed and it is suggestive from the figures that miners, metal workers, and spinners and weavers have a higher than average incidence of blindness.

When traumatic cases are omitted there appears to be no detectable difference in the relative total incidence of blindness in the sexes.

The findings of the present analysis compared with the results given by Sorsby for England and Wales are given below[2]. Sorsby's figures relate to the following investigations and reports:

Prevention of Blindness Committee (1938).

Northern Counties Association for the Blind (1938).

Counties of London and Middlesex (Sorsby 1943).

For England and Wales 77·2 per cent. of the registered blind in 1943 were over 50 years of age; 20·8 per cent. fell in the age group 16-50 years, and 2·0 per cent. in that of 0-16 years. The comparative figures for Scotland are :—71·1 per cent. at 50 and over, 25·7 per cent. in the 16-49 age group, and 3·2 per cent. in the group 0-15 years.

Causes of Blindness	All Scotland	Prevention of Blindness Committee	Northern Counties	London and Middlesex
	7297 cases	5290 cases	10,000	9,562
	per cent	per cent.	per cent.	per cent.
Cataract	17·1	25	33·6	21·2
Myopia ...	14·9	10·2	8·5	12·1
Glaucoma ...	8·1	11·8	10·0	14·0
Defects of congenital hereditary and developmental origin ...	9·4	11·0	7·0	10·4

It is probable that some of the considerable differences in percentage are due to different methods of classification. In Scotland, *e.g.*, myopia has been noted as the main disease causing blindness when complicated by cataract. This may explain the higher incidence of myopia and the lower incidence of cataract, but an attempt at explanation can only be conjecture at present, and further statistics may make the comparison of figures more reliable.

Several difficulties have been encountered in compiling the tables, and a scheme for the annual publication of blindness statistics by the Statutory Department has been suggested.

Acknowledgments. The tables were compiled by the Statistical Branch of the Department of Health for Scotland and much patience and expert knowledge were required to overcome many difficulties.

Particular thanks are due to the head of that Branch for invaluable collaboration and advice without which the publication of this report would not have been possible.

The comprehensive report for Glasgow and South-West Scotland was kindly placed at our disposal by the authors, Dr. John Marshall and Dr. H. E. Seiler.

The report was submitted to the Sub-Committee on Prevention of Blindness of the Secretary of State's Scientific Advisory Committee and is published with their approval.

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LOCAL TREATMENT OF INTRABULBAR INFECTIONS*†

6. Treatment of Staphylococcuspanophthalmia by Intrabulbar Injection of Penicillin

BY

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As shown in previous articles (Roenne[1-5]) artificial intrabulbar infections can be cured by injecting chloramine-T into the vitreous body. This treatment has, however, only limited effect, as an inoculum of more than 5,000 microbes is difficult to sterilize. This being the case the testing of other bactericides was desirable, principally the sulphonamides and penicillin. From preliminary experiments it was found that sulphonamides, in conformity with the reports of other workers, have but a very slight effect, whereas penicillin proved to be particularly efficacious. Even with weak concentrations of 1-2 Oxford Units, the effect on infections in the vitreous humour was considerable. The effectivity improved with increasing concentrations of penicillin, even an inoculum of 5,000,000 virulent staphylococci was overcome and the eye preserved. It was also observed, that penicillin injected into the vitreous was extremely durable; it disappears so slowly that a bacteriostatic concentration is preserved for many hours, up to 48, all depending on the original quantity of penicillin (Roenne[4-7]).

American workers have also taken an interest in the treatment of intrabulbar infections with penicillin. von Sallmann, for instance, tested the effectiveness of treatment of infections induced by pneumococci and staphylococci injected into the anterior chamber.

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Treatment was partly by means of drop instillation and partly by iontophoresis. In the absence of any lesion of the lens both forms of treatment gave good results. In the case of a lens-lesion the result was inferior, though the effect was still apparent. Infections produced by clostridium Welchii did not respond to treatment.

von Sallmann, Meyer and Di Grandi demonstrated that neither iontophoresis nor general treatment had any effect on inflammation in the vitreous body, and therefore they tried injecting penicillin direct into the vitreous, with good results. They observed that intravitreal injections of penicillin produced lesions of the retina at the site of the puncture and opacities and sometimes haemorrhage in the vitreous body. The present author has previously published a short series of experiments on the toxicity of penicillin injected into the vitreous and has shown that penicillin concentrations of 10-100 O.U. do not produce definite pathological changes (Roenne[4]), whereas, the opposite is the case with sulphonamides in the necessary concentrations (Leopold and Scheie). von Sallmann *et al.*, demonstrated that for 24 hours after the injection of penicillin it was possible to detect a bacteriostatic penicillin concentration in the vitreous.

According to Struble and Bellows, demonstrable quantities of penicillin are found in the vitreous after subconjunctival injections. Leopold's investigation gave the opposite result. He found, that only when there was inflammation in the anterior section of the eye, could a therapeutic penicillin concentration be obtained. When penicillin was injected into the anterior chamber, Leopold found a therapeutic concentration in the vitreous, and the concentration became higher if there was inflammation in the anterior chamber or if iridectomy had previously been performed. According to Leopold, intramuscular and intravenous treatment does not lead to therapeutic penicillin concentration in the vitreous. In accordance with this he found that intravenous injection as a means of treating intravitreal infection is of no value. Subconjunctival injection and injection into the anterior chamber could be protective against suppuration, but penicillin injections into the vitreous were most effective. All eyes that were given over 1,000 O.U. in the vitreous were practically normal. With decreasing amounts of penicillin the results became poorer, but even 5 O.U. showed some effect. In all cases Leopold employed an inoculum of about 400 microbes of a haemolytic staphylococcus aureus.

Toxic effect of penicillin on the eye

With the exception of slight opacities, clinically observable toxic phenomena are not occasioned by injection into the vitreous of even very considerable quantities of penicillin viz : 100-500 O.U.

Similar opacities occur after the injection of saline in various concentrations. Whether penicillin in therapeutic concentrations produces histological changes, cannot be decided at the present stage with absolute certainty, particularly because the preparations available to the writer were not in a highly purified state. Nevertheless, it seems justifiable to say now, that even fairly large doses of 100-500 O.U. of penicillin are practically harmless to the eye.

Penicillin treatment of infections in the vitreous body

Technique.—The rabbits employed weighed about 2,000 grammes. They were inoculated with a highly virulent staphylococcus aureus strain, which, with an inoculum of 50 bacteria led to perforation of the eye in less than four days. Even with an inoculum of 5 bacteria there were 100 per cent. takes. (For details, see previous works (Roenne[2 3 5 6 8])). The penicillin, which was supplied by Professor K. A. Jensen and produced by him, was standardized according to the usual principles. During the course of the investigation, preparations of penicillin purified in various ways were employed. Nevertheless, the various preparations all had identical effects. In every experiment 0·2 c.c. of penicillin was injected into the vitreous.

The inoculation by intravitreal injection with various inocula followed a logarithmic scale, viz: 5, 50, 500, ———, 5,000,000 microbes; after a certain interval, the eyes were treated with penicillin injected into the vitreous.

As will be seen from Table I, there were four experimental groups, treated respectively with 0·2, 2, 20 and 200 O.U. The first column shows the amount of penicillin in O.U., the second column the interval between inoculation and treatment, and the next columns show results of the treatment with increasing inoculum. In these columns, the first cipher gives the number of cured eyes, the second cipher the number of those not cured. For each group the tests in which the number of cures was lower than 100 per cent. but higher than *nil* are circumscribed. The experiments within these limits correspond roughly to 50 per cent. cures. It is significant that an increase in the penicillin concentration improves the results, and greater quantities of bacteria can be destroyed.

To obtain a more concise picture of the effects of the treatment, we must know the quantities of bacteria present in the eye at the moment when the penicillin is injected. The following method was adopted: In a previous paper, (Roenne[6] Fig. 2) the growth curve of staphylococci in the eye was calculated for two different inocula, 5,000,000 and 50 bacteria respectively. If the ordinate is the logarithm of the concentration of bacteria and the abscissa is the experimental time in hours, we can, by the method of least squares, obtain the following equations for the bacterial growth. With an

TABLE I

Penicillin injected after inoculation

Influence of size of inoculum, penicillin concentration and interval between inoculation and treatment upon the therapeutic result

Groups	Dose of penicillin injected	Interval in hours	Inoculum 50	500	5,000	50,000	500,000	5,000,000
I	0.2 O.U.	1	2-0	2-0	1-1	0-2	0-2	–
		2	3-1	2-2	1-3	0-4	0-2	–
		4	2-0	0-2	0-2	0-4	–	–
II	2 O.U.	1	–	–	–	2-0	2-0	1-1
		2	–	–	–	2-0	2-0	1-1
		4	–	–	–	2-0	3-1	2-2
		8	–	–	2-0	2-0	0-2	0-2
		16	2-0	2-0	2-0	0-2	0-2	–
		24	1-3	0-4	0-4	–	–	–
III	20 O.U.	1	2-0	2-0	2-0	2-0	2-0	1-1
		2	2-0	2-0	2-0	2-0	2-0	1-1
		4	2-0	2-0	2-0	2-0	2-0	1-1
		8	2-0	2-0	2-0	2-0	1-1	0-2
		16	4-0	4-0	3-1	2-2	0-4	0-2
		24	4-0	2-2	0-4	0-4	0-2	0-2
		32	0-4	0-4	–	–	–	–
IV	200 O.U.	1	–	–	–	–	2-0	2-0
		2	–	–	–	–	2-0	2-0
		4	–	–	–	2-0	2-0	0-2
		8	–	–	2-0	2-0	1-1	1-1
		16	2-0	2-0	2-0	2-0	1-1	0-2	0-2
		24	2-0	2-0	1-1	0-2	0-2	–	–
		32	2-0	1-1	0-2	0-2	0-2	–	–

inoculum of 5,000,000 bacteria: $y = 0{\cdot}28x + 5{\cdot}44 \pm 0{\cdot}21$, and for an inoculum of 50 bacteria: $y = 0{\cdot}26x + 0{\cdot}11 \pm 0{\cdot}31$. This means that the growth velocity in the two experiments is the same (the same coefficient of inclination). As the distance along the ordinate between the growth curves is 5·43 (the theoretical value is 5·00), with a mean error of ± 0·36, the conformity cannot be much better. Therefore it must be justifiable to interpolate the growth curves for other inocula. The writer chose the experiment with 5,000,000 bacteria to determine the point of intersection with the ordinate-axis, because that experiment embodied the greater number of observations.

TABLE II

Calculated values for the logarithm of the bacteria concentration with various inocula after different times

Inoculum	Hours after inoculation							
	1	2	4	8	16	24	32	48
5	—	0·1	0·6	1·7	3·9	6·2	8·4	—
50	0·7	1·0	1·6	2·7	4·9	7·2	—	—
500	1·7	2·0	2·6	3·7	5·9	8·2	—	—
5,000	2·7	3·0	3·6	4·7	6·9	—	—	—
50,000	3·7	4·0	4·6	5·7	7·9	—	—	—
500,000	4·7	5·0	5·6	6·7	—	—	—	—
5,000,000	5·7	6·0	6·6	7·7	—	—	—	—

Growth curves for all inocula employed in the present experiment having been drawn, the logarithm of the bacterial concentration at the various points of time can be read direct, and the values found are shown in Table II. As the bacterial concentration per c.c. in the eye does not rise beyond 8·5 logarithmic measurement, no values higher than this are included in the table. Then, within each series of experiments (Table I), we determine with which inoculum we have 50 per cent. cured, and by means of Table II we can ascertain directly the corresponding concentration of bacteria. As will be understood, this determination rests on a

rather rough estimate. The calculation is completed for all experimental series and groups and the result is shown in Table III.

TABLE III

The logarithm of the bacterial concentration for 50 per cent. cured at the time of treatment.

Penicillin concentration	Inoculum 5	50	500	5000	50000	500000	5000000
0·2 O.U.	0·6	1·0	1·7	—	—	—	—
2 O.U	5·6	6·0	7·0	5·8	5·1	5·3	5·7
20 O.U.	7·3	7·2	6·5	6·9	5·7	5·6	6·3
200 O.U.	8·5	8·5	8·2	8·0	7·9	6·7	7·7

From this table it will be seen that within each group (penicillin concentration) there is surprising constancy in the bacterial concentration that corresponds to 50 per cent. cured. This implies that it is more or less immaterial whether the quantity of bacteria present has been produced by a large inoculum which has grown within a few hours, or a small one with a long period of growth.

This is rather extraordinary, as one would expect *a priori* that in the case of an infection 16 to 24 hours old, the microbes would have the opportunity of spreading among the tissues of the eye, and in many places they would be well sheltered from the attacking substance. The result we have observed means, presumably, that the penicillin is distributed very readily in the eye tissues and everywhere attains a considerable bactericidal concentration, and that this concentration is maintained for a sufficient length of time.

Velocity of decrease in penicillin

Table IV represents a summary of a number of experiments performed in exactly the same manner as those in Table I, only with the one difference, that the penicillin was injected *before* the eyes were inoculated. The table is in principle similar to Table I, and here again the experiments in which the number of cures was less than 100 per cent. and over *nil* are circumscribed for each group. Even a casual examination shows that these delimited experimental values (except in the case of Group I) form very nearly a straight line within each group, and that the three lines thus arrived at are almost parallel.

TABLE IV

Penicillin injected before inoculation

Influence of size of inoculation, penicillin concentration and interval between inoculation and treatment upon the therapeutic result

Groups	Dose of penicillin injected	Interval in hours	Inoculum 5	50	500	5,000	50,000	500,000	5,000,000	Calculated penicillin concentration in O U at time of inoculation
I	0.2 O U.	0	2-0	2-0	1-1	0-2	0-2	0-2	0-2	0 2
		1	4-0	2-2	1-3	0-4	0-4	0-2	0-2	
II	2 O.U	1	-	-	-	-	-	-	-	
		2	-	-	-	-	2-0	1-1	1-1	
		4	-	-	-	2-0	1-1	1-1	0-2	1
		8	-	2-0	2-0	2-0	1-1	0-2	0-2	0·6
		16	2-0	4-0	3-3	2-4	0-4	0-4	0-2	0 2
		24	0-2	0-2	0-2	-	-	-	-	0 06
III	20 O.U.	1	-	-	-	-	-	-	-	
		2	-	-	-	-	-	-	-	
		4	-	-	-	-	2-0	2-0	2-0	11
		8	-	-	-	2-0	1-3	4-0	3-1	6
		16	2-0	2-0	2-0	4-0	3-1	3-1	2-2	2
		24	4-0	6-0	4-0	4-2	2-4	0-2	0-2	0 6
		32	4-0	4-0	3-1	0-4	0-4	-	-	0·2
IV	200 O.U.	1	-	-	-	-	-	-	-	
		2	-	-	-	-	-	-	-	
		4	-	-	-	-	-	-	-	
		8	-	-	-	-	-	2-0	2-0	64
		16	-	-	-	-	2-0	2-0	2-0	20
		24	2-0	2-0	2-0	2-0	2-0	2-0	1-1	6
		32	2-0	2-0	2-0	2-0	4-0	3-1	2-2	2
		48	2-0	4-0	3-1	3-1	1-3	0-4	0-2	0·2
		56	1-1	0-2	0-2	-	-	-	-	0·06

This becomes still more obvious if we plot the results in a coordinate system in which the axes are the logarithm of the inoculum (log. c) and the interval between the penicillin injection and inoculation (t) respectively. Thus, it is obvious that there is a rectilinear relation between the interval between penicillin injection and inoculation (t) on the one hand and the logarithm of the bacterial concentration (the concentration that is cured in 50 per cent. of cases) on the other; briefly expressed: $t = a \log c +$ constant. As, moreover, it is obvious that the quantity of penicillin present at the moment of inoculation must be a function of t (the interval between the penicillin injection and the inoculation), the natural assumption is, that the inclination of the three curves is an expression of the relation between the penicillin concentration at the moment of inoculation and the bacterial dose (50 per cent. cured).

Furthermore, according to the laws of diffusion and osmosis, we must assume that the disappearance of the penicillin from the eye owing to diffusion, absorption and destruction, proceeds logarithmically, *i.e.*, that a certain percentage of the penicillin disappears per time unit; the result of these processes must be that the relation penicillin-concentration—bacteria-concentration is reproducible by a straight line in a double logarithmic coordinate system.

Returning to Table IV, we find that the distance between the two pairs of the three parallel lines along the Y-axis (the interval between penicillin injection and inoculation) is 16 hours. (As stated, in the first experimental group, the material is too small for the calculation of a line analogous to the other groups, but the one point in this group, which we can make use of, agrees with the other observations). The difference in the penicillin concentration between each experimental group is 1 : 10. This signifies that in the course of 16 hours, the original penicillin concentration has fallen to 1/10, in the course of 32 hours to 1/100 and in 48 hours to 1/1,000, which again shows the correctness of the previous assumption, that the penicillin in the eye disappears according to a logarithmic curve.

On this basis, for each experimental group, we can calculate the penicillin concentration present at any moment. The values found are shown in the last column of the table. (*)

Ratio of penicillin concentration and bacteria concentration

We now proceed to compare the calculated penicillin concentrations with the appropriate inocula (log c) and plot the values

* Naturally, this determination is easier to carry out by a direct analysis of the penicillin content of the eye at various times after injection. An analysis of this kind was made and it was found that the halving time, *i.e.*, the time at which half of the penicillin has disappeared, is just over 5 hours. The method described above gives a halving time of just under 5 hours, so that there is a satisfactory agreement between the two.

found in a coordinate system in which the axes are the logarithm of the bacteria concentration and the logarithm of the penicillin concentration.

TABLE V

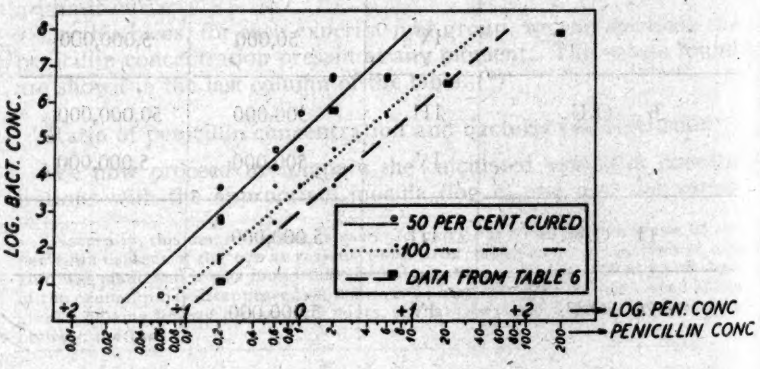
The number of bacteria destroyed in 100 and 50 per cent of cases with various penicillin concentration

Calculated penicillin concentration	Group	Cured	
		100% cured	50% cured
0·06 O.U.	II	—	—
	IV	—	5
0·2 O.U.	I	50	500
	II	50	500
	III	50	500
	IV	50	5,000
0·6 O.U.	II	5,000	50,000
	IV	500	5,000
1 O.U.	II	5,000	500,000
2 O.U.	III	5,000	5,000,000
	IV	50,000	5,000,000
6 O.U.	III	500,000	50,000,000
	IV	500,000	5,000,000
11 O.U.	III	5,000,000	—
20 O.U.	IV	5,000,000	—

On the surface, it might seem that we are here begging the question. Actually, of course, this is not the case. Really, all we have done is to replace the original ordinate values in each experimental group (for each line in the coordinate system) with new values, *i.e.*, the time with the logarithm of the penicillin concentration and this does not alter the shape of the curves. Let the unit of the new coordinate system correspond to 16 hours in the old system and the inclination of the lines is exactly the same.

With Table IV as basis, Table V is formed by comparing the calculated penicillin concentrations with the inocula which can be overcome in respectively 100 and 50 per cent. of cases. The first column gives the concentration of penicillin at the time of inoculation and the second column the respective experimental groups. The third and fourth columns give the quantity of bacteria destroyed for 100 and 50 per cent. respectively. An interpolation has been made because, as stated, we have not exactly 50 per cent. cures in all experiments, but sometimes more and sometimes less. The correlative values thus arrived at are plotted in a coordinate system (Fig. I) for 100 and 50 per cent. cures. It appears from the figure that they are very nearly linear functions, as was to be expected from the theoretical considerations. From the curves, we can now read directly how high a concentration of penicillin is necessary to destroy or sterilize a certain quantity of bacteria in 100 and 50 per cent. of the experiments. It is of great importance in the practical therapeutic employment of penicillin, that ten times the concentration of the bacteria requires only about twice the penicillin concentration, or conversely, that ten times the penicillin concentration increases the number of bacteria that can be destroyed a thousandfold.

However, the numbers in the figure representing the penicillin concentration are not absolute, but merely relative values. For example, if the quantity injected into the eye in a given case is



2 O.U., the concentration per c.c. will not be 2 O.U. but somewhat lower, because the volume of the rabbit eye is more than 1 c.c. As a rule we must reckon with a dilution of about 2-3 times, so that with this quantity of penicillin the concentration in the eye is 0·5-1 O.U. This, of course, involves a parallel displacement of the curve, but no change in its inclination.

In addition, Table III shows quite distinctly, that an increase of the penicillin dose involves an increase in the number of bacteria that can be affected by the treatment. On the basis of Table III, we can calculate the mean value of the bacterial concentration capable of being killed in 50 per cent. of cases by the penicillin concentrations employed, and we find the values shown in Table VI, column 2. In column 3 are the numbers of bacteria which similar doses of penicillin, according to Fig. 1 (Table IV) can deal with. As appears from Table VI, there is a rather considerable difference between the two main experiments Penicillin treatment undertaken *after* the inoculation gives a much inferior therapeutic result to that obtained from treatment *before* the inoculation. Table III showed that primarily the result of the treatment depends only on the concentration of bacteria, and is independent of the interval between inoculation and treatment. It might perhaps be natural to assume that this would also be the case when the interval was "negative," *i.e.*, when the penicillin treatment was undertaken before the inoculation in which case the figures in column 2 and 3 would be similar. As Table VI shows, this is not the case, and must be due to special circumstances. In the first place we must bear in mind the radical change that takes place in the biology of the bacteria when they are transferred from the nutritive media to the eye, and it is a natural assumption that by this means the vitality of the microbes is lowered very considerably for a time, that many of them die and that the survivors become acclimatized to their new conditions of life only after a certain time has passed. Indeed, it is a fact, that quantitative tests of bacterial growth in the eye show a heavy initial fall in the number of bacteria immediately after inoculation, and only reaches its original size after some hours (Roenne[4]). Thus, the difference in the results of the treatment in the two experimental groups may be explained by the decrease in vitality of the bacteria at the moment of inoculation.

If the relation penicillinconcentration—bacteriaconcentration for the experiments in Table IV can be expressed by a linear function in a double logarithmic co-ordinate system, this, of course, should also apply to the experiments in Table I. It appears, however, from Table VI, column 2 that these experimental data only with difficulty compare favourably with this hypothesis. However, it must be borne in mind that even if the figures in Table III, column 2 are based upon a fairly considerable material, in contrast to

TABLE VI

*Comparison between treatment before and after inoculation. Logarithm of the number of bacteria destroyed in 50 per cent. of cases.

Penicillin concentration	Data from Table 3	Data from 50% graph in Fig 1
0·2 O.U.	1·1	3·1
2 O.U.	5·8	5·9
20 O.U.	6·5	8·7
200 O.U.	7·9	11·5

*It should be noted that the last two values in Table 6 col. 3 are extrapolations and it is therefore uncertain whether we can reckon with these values as true. It is not impossible that the linear function does not apply to extremes.

Table IV there are so many more approximations and interpolations that they are encumbered with very considerable uncertainty. Without embarking upon an exposition of the error of these values (indeed, it cannot be calculated exactly), it may be stated that within the bounds of probability, the error is of such an order that these experiments also follow the general relation for the penicillin-concentration—bacteria concentration.

With these reservations, it is nevertheless possible, with only four values, to draw the straight line representing the function penicillin-concentration—bacteria concentration for the experiments in Table I. This line is also plotted on Fig. 1 and is practically parallel with the two already referred to. Of course, here again we must remember the dilution that takes place in the eye.

Discussion

Although several approximations and interpolations have been used in the arrangement of the basic experimental data, the parallelism of the graphs in Fig. 1 can hardly be accidental, but must be the expression of a biological law. Besides, the experiments are a further example of the remarkable bactericidal property of penicillin and show, moreover, that it is practically unlimited how intense infections it is possible to cure when sufficiently large doses are employed.

Finally, on going into the question of how this relation between

quantity of bacteria and penicillin concentration compares with our experience in other directions, we find that there is reasonable conformity. With general penicillin treatment of, for example, septicaemia, the penicillin concentration obtained in blood and tissues fluid is of a maximum average of one-fifth to one-third O.U. per c.c. On looking at the curve (the one based on the experiment in Table I), we find that it corresponds to a bacterial dose of about 10-200 microbes per c.c. On correcting for the aforesaid dilution error, the quantity of bacteria susceptible to treatment turns out to be 100-1,000 per c.c. This of course is much higher than the concentration of bacteria in the blood of sepsis patients, but the difference does not seem to be greater than is permissible, especially when we realize that direct comparison is impossible. The explanation is that the time factor, *i.e.*, the time during which the microbes are exposed to a penicillin concentration that will injure or inhibit them, undoubtedly differs in the clinic and in these experiments. In the clinic, the effective penicillin concentration is maintained from for many hours to several days, whereas in the experiments it was presumably maintained only some few hours, at any rate when the quantities of penicillin were small.

Unfortunately, in these experiments it is impossible to derive any direct information as to the time factor and its importance to the result of the treatment, because both this and the penicillin dose enter into the experiments in such a manner, that the effects of the two different factors defy analysis. But it is obvious that investigations into the importance of the influence of the time factor on the result of the treatment is a question of great interest, not only theoretically but also clinically. For example, it is possible that in the case of penicillin too, the protracted continuous treatment with the appertaining low blood concentrations can—and will—be replaced by a therapy which brings the blood and tissue concentrations up to high values for a short time.

Although these investigations have been concerned only with a single form of bacterium, it is reasonable to suppose that the same laws will apply to the action of penicillin on other microbes, even if in that case the constants of the equation will be altered. For the present it is justifiable to assume that in penicillin we possess the best weapon so far at our disposal for combating intra-ocular infections, and that by local treatment we obtain a number of advantages not possible with general treatment viz: (1) the maintenance of a bactericidal concentration of penicillin for a long period, 16-24 hours; (2) only small quantities of penicillin are necessary; (3) Leopold and Struble and Bellows have shown that general penicillin treatment does not lead to any demonstrable concentration in the vitreous or at any rate only in very small quantities. The present author has made a series of similar experiments and can verify

the results. With very large doses of 5,000 O.U. per kg. one can obtain concentrations of about 0·05-0·10 O.U. per c.c. in the vitreous.

Thus, there can hardly be any doubt, that for the moment local treatment—injection of penicillin in the vitreous humour—is the most rational therapy for intra-ocular and especially intra-vitreal infections. Chloramine-T previously employed by the writer (Roenne[1 2]) is much less effective, partly because of its toxic action when in large concentrations, partly because the bactericidal effect is maintained only a few hours.

The only drawback of penicillin is its specificity. Fortunately it attacks most of the microbes we encounter in infected perforating lesions in human pathology.

Conclusions

(1) In therapeutic concentrations of 100-500 O.U. the injection of penicillin into the vitreous body is practically harmless.

(2) Intra-vitreal penicillin treatment of infections in the vitreous body is extraordinarily effective compared with other forms of application and other substances.

(3) The effectivity of the treatment is directly proportional to the concentration of penicillin.

(4) Within fairly wide limits the result of the treatment is dependent only on the number of bacteria in the eye and not on the interval between inoculation and treatment.

(5) The method employed provides indirect information as to the velocity of the absorption of the penicillin from the vitreous body. The values found agree very well with the results gained by direct bio analysis.

(6) The velocity of the absorption of penicillin from the vitreous body is expressed by the halving time, about 6 hours.

(7) The function: log bacteria-concentration—log therapeutic penicillin-concentration, is of first degree.

(8) For intrabulbar, especially intravitreal inflammation, the injection of penicillin into the vitreous body is the best therapy at the moment.

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RETINAL DETACHMENT DUE TO WAR TRAUMA

BY

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THIS enquiry was made to find out the incidence of retinal detachment directly attributable to the effects of war missiles, the relation of this incidence to other types of retinal detachment occurring in soldiers serving in a field force, and the comparative results of treatment and prognosis.

In the 1914-18 war Gonin's principle of locating a retinal tear or tears accurately and sealing these by diathermy with subsequent drainage of the inter-retinal fluid was not known or practised and so in the 1939-45 war an opportunity arose to try out modern surgical treatment in such cases as seemed to offer a reasonable chance of effecting some improvement.

This work was done in the M.E.F. 1941-43 and the B.L.A. 1944-45 where to the 15th (Scottish) General Hospital in Egypt, the 102nd Field Hospital in Normandy and the 108th General Hospital in Belgium the majority of retinal detachment cases and serious ocular and orbital wounds with retained foreign bodies were referred.

In the M.E.F. 78 cases of retinal detachment were treated and in the B.L.A. 18, making a total of 96. I have classified these cases as follows:—

TABLE A

	M.E.F.	B.L.A.	Totals
Trauma (Military)	18	2	20
Trauma (Civil)	23	3	26
Choroido-retinal degeneration ...	21	5	26
Cystic degeneration	13	3*	16
Myopia	3	5	8
	78	18	96

* One of these had both eyes operated on

(Included in the classification cystic degeneration were 12 cases in which part of the detachment appeared cystic. In 9 of these around the base of the cyst there was a well defined arc of pigmentary degeneration in the choroid and retina at the posterior periphery of the cyst up to the ora serrata where these changes ceased. In 3 others these changes were faint. The ophthalmoscopic appearance suggested a line of adhesion between the retina and choroid which was more marked in some cases than in others. In one case these changes were present in only part of the arc at the posterior periphery of the cyst. A retinal tear or tears were found in all 12 cases and 7 of these showed multiple tears. In 2 cases a sudden spread of the detachment from the limits of the cystic area caused separation of the macula).

It will be seen from Table A. that approximately 20 per cent. of all types of retinal detachment occurring in a field force were due to trauma from military missiles.

The incidence of retinal detachment due to military trauma among other battle casualties is impossible to assess with accuracy for in the M.E.F. the wounded were distributed to at least 9 General Hospitals with eye surgeons and in some instances went through as many as 4 of these. There was therefore in the early days of the Western desert campaign a multiplication of statistics which was confusing enough, and this was increased by security measures

which made it impossible for Medical H.Q. to disclose information about the numbers of wounded. There were 475 battle casualties admitted to the eye department of the 15th (Scottish) General Hospital beginning with General Cunningham's offensive in 1941 and ending with the conquest of North Africa in May, 1943. Of these 18 had retinal detachment due to military trauma. But this figure is misleading for many of the cases sent to this hospital were selected.

In the B.L.A. precision in this matter was also rendered impossible by the rapid evacuation of wounded by sea and air in the early stages of the Normandy campaign and indeed until early September, 1944, we were seldom allowed to retain any but a few special cases for more than 2 or 3 days. There were only 2 cases of retinal detachment due to military trauma.

So all that may be said is an impression that retinal detachment due to war missiles in which the eye is left reasonably intact and has not a large penetrating wound nor is collapsed or so grossly disorganized as to make excision necessary is a rarity in the wounded.

Of all types of retinal detachments classified above 13 were officers—one of these a nursing officer Q.A.I.M.N.S.(R.); 16 were British other ranks, Royal Navy 2 cases, Royal Air Force 2 cases, Royal Canadian Air Force 1 case, Royal Canadian Engineers 1, Canadian Infantry 1 and Merchant Navy 1 case.

The nationalities were British 76, Canadian 3, New Zealand 1, South African 3, Indian 3, Greek 4, Polish 2, Czech 1, Yugo-Slav 1, German 1 and Italian 1

Table B. shows the age incidence of the various types of detachment.

TABLE B

Age (years)	Trauma Military	Trauma Civil	Choroido-retinal degeneration	Cystic degeneration	Myopia
20–30	16	21	21	10	3
31–40	4	5	3	5	2
41–47	—	—	2	1	1
	20	26	26	16	6

As might be expected the highest incidence of retinal detachment, particularly that due to military trauma and the types of

trauma seen in civil practice is in young soldiers in the third decade of life for these form the majority of a field force.

Analysis of cases of military trauma. Twenty cases

The area of retinal detachment was extensive in a number of these cases. Large splashes of blood on the retina, in the choroid and vitreous, exudates, subsequent fibrous organization of intra-ocular blood, the presence of blood between the retina and the choroid, and the lowered intra-ocular pressure as the result of severe concussion of the eye from blast were factors against successful treatment. It is probable that vitreous gel passes through some of the large ragged rents in the retina and is a mechanical factor in preventing choroido-retinal adhesions from forming after diathermy.

In one case in this series of 20 the intra-ocular changes were such that surgical operation seemed to hold out no hope of effecting any improvement. The soldier had sustained a blast injury from an aerial bomb which exploded 200 yards to his right front. Three months after the injury the retina was totally detached; dense white broad sheets of organized blood clot were adherent to it in the lower half of the eye, over the macula, in the upper nasal quadrant, and in front of the equator above. The inferior branches of the central retinal vessels immediately below the optic disc were kinked and tortuous. There was much pigmentary disturbance in the retina, and the intra-ocular pressure was 6.5 mm.Hg. Vitreous had herniated forwards into a coloboma of the iris in the lower temporal quadrant, the suspensory ligament having been ruptured at this site.

Spontaneous recovery occurred in 2 officers. One of these, Lieut. C. McL. S., aged 23 years, a Royal Engineer, was affected in his right eye by the blast of a land-mine explosion on October 20, 1944, in Belgium. Three quadrants of the retina were detached, only the upper nasal quadrant remained in place and a corresponding part of the lower temporal field was present. In the upper temporal quadrant the retina was ballooned forwards and the macula was detached. There was a faint " sunflower " opacity in the posterior lens cortex. The patient was wearing - 5·5 D.sph. The right and injured eye was further complicated by the effects of an old injury. In April, 1943, an explosion of gun-cotton had blown a foreign body into this eye. This foreign body was oval in shape, about 1·5 mm. long, lay in the 6 o'clock meridian near to the ora serrata. The end near the ora serrata was free and that nearer the equator attached to the retina by a fibrous strand, and on this anchorage it floated in the vitreous and changed position on postural alterations of the eye and head. This foreign body was non-magnetic. The wound of entry was not evident. A scarred area of retina and choroid below the macula and near the

equator, from which greyish and white tags of fibrous tissue projected into the vitreous, suggested that the foreign body may have struck the fundus at this site and rebounded into the vitreous. Some vitreous opacities, a few of which were coarse, lay in front of the equator in the lower temporal quadrant. Some red blood cells could be seen with the slit-lamp in the anterior part of the vitreous. Repeated examinations in different postures revealed no hole or split in the retina at any time. At first I thought that the fibrous bands passing from the retina into the vitreous and the presence of the intra-ocular foreign body might operate against successful reposition of the retina.

The patient was confined to bed, lying on 2 pillows, the right eye was atropinized and both eyes occluded by pads and a binocular bandage. On the seventh day after the injury the retina in the upper temporal quadrant was in place. His position was then changed to sitting and during the next 14 days the retina was replaced over the macular area and then to the equator and slightly beyond in the lower quadrants of the eye. Twenty-two days after the injury a shallow residual detachment remained near the ora serrata around the site of the intra-ocular foreign body between the 5 and 7 o'clock meridian. He was then allowed to wear stenopaic glasses. For some 10 more days there was little change in this area of detachment then within 7 days the inter-retinal fluid absorbed and on November 28, 1944, 39 days after injury the retina became replaced and the visual field fully restored and right vision 6/18. On December 6, 1944, vision was 6/12 with glasses correcting the myopia (– 5·5 D.sph.). Later this became 6/6 despite the early posterior cortical lens changes, the probable sequelae of the 1943 injury. Follow-up examinations up to June 21, 1945, when this officer was last seen in Belgium, showed the retina completely in place, the visual field full and the visual acuity maintained at 6/6 with – 5·5 D.sph.

This case affords several useful lessons. (1) That in blast injuries where there is no tear or split expectant treatment is justifiable. (2) That the visual results after reposition of detachment of the macula are good where there is no retinal tear, no pathological lesion at the macula and there has been no operative treatment and the eye has been treated expectantly.

Spontaneous recovery also occurred in another officer, Major A. D., aged 29 years, who was wounded by a fragment of shell which struck the temporal end of his left supra-orbital margin whilst he was reconnoitring the position of some German tanks on an escarpment in the Western desert. His head was exposed above the tank turret. There were a severe contusion of the left eye and a rupture of the choroid at the macula, and large splashes of blood and exudate were present on the retina and in the choroid

and vitreous of the upper temporal quadrant. One month after this wound the retina in the upper temporal quadrant became detached. There was no hole evident. The intra-ocular pressure was 6 mm.Hg and visual acuity less than 6/60. Postural treatment was tried for three and a half weeks. The detached area became slightly smaller and some of the inter-retinal fluid was absorbed. Within two months of the onset of the detachment the retina was completely restored to its normal place.

In this group of 20 cases 3 had lost one eye (the left in each case) which was destroyed beyond hope of repair at the time of wounding. In 9 cases the right was affected, and in the 11th the retina was detached in the left eye. In most the actual duration of the retinal detachment was difficult to assess accurately. In some instances it was apparent a few days after wounding; in others it was obscured by intra-ocular haemorrhage and did not become evident for several weeks, while in a few it occurred as a late sequel, 6 to 14 months after the injury. In the latter the contracture of fibrous tissue attached to the retina and the lowered intra-ocular pressure were likely causes. The times, taken from the date of wounding, were: 1 to 11 days, 7 cases; 1 to 4 months, 9 cases; 4 to 6 months, 3 cases; and 14 months, 1 case.

Retinal detachment occurred in association with an intra-ocular foreign body in 5 cases. The detachment happened immediately in one case, 53 days later in another, and in a third 6 weeks after the extraction of an intra-ocular foreign body, at another hospital, by the posterior route, in which diathermy was not used at the site of the sclerotomy. In the other 2 cases the date of onset of the retinal detachment was not known. Of the intra-ocular foreign bodies, three were extracted by the magnet through the posterior (scleral) route, one was removed by a snare and forceps guided under ophthalmoscopic view, and another was too small and non-magnetic, and so was left.

In 3 cases tangential cuts in the sclera were caused by a shell fragment, which did not remain in the eye but passed on into the orbital tissues. Blast from a shell, a mortar bomb and an aerial bomb caused retinal detachment in 4 cases; hand grenades were responsible for 5, land-mines for 2, a rifle-butt for 1, a bullet traversing the orbit for 1, and a fragment of mortar bomb for 1.

The site of the retinal detachment was total in 2 cases, almost total in 2, involved the lower half of the retina in 5, the upper and lower temporal quadrants in 2, the upper and lower temporal quadrants and lower nasal quadrant in 5, the upper and lower nasal quadrants and the lower temporal quadrant in 2, the upper temporal quadrant in 1, and a curtain-like detachment affecting the upper nasal and upper temporal quadrants and the lower nasal and lower temporal quadrants in 1 (see Fig. 1).* There was no

* The blocks for Figs. 1, 2, and 3 have been lent by the Editor of the *Brit. Med. Jl.* to whom my best thanks are due.

hole present in this case. Fig. 2 is a diagram of the operation. The internal rectus muscle was divided temporarily at its insertion in front of two mattress sutures. Beneath the muscle belly was seen a tangential wound in the sclera just in front of the equator in the

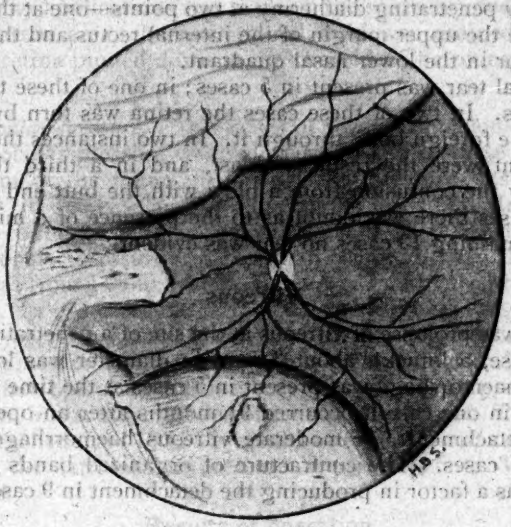

FIG. 1.

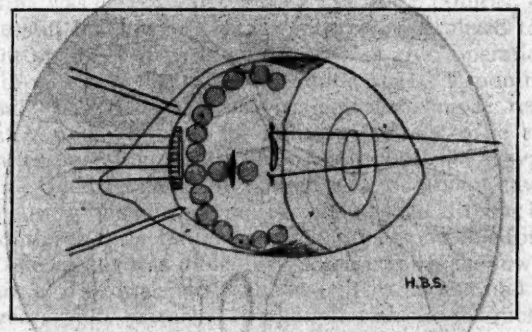

FIG. 2.

9 o'clock meridian. The wound was linear, 3 mm. long, vertical and gaping; it revealed the choroid. The application of surface diathermy on either side of it closed it by contracting the sclera. A line of surface diathermy application was made at the equator

between the nasal borders of the superior and inferior rectus muscles. From the upper and lower ends of this line applications of diathermy were made forwards to the ora serrata. Thus a frame was made just clear of the scar tissue inside the eye and with the object of pinning down the retina. The inter-retinal fluid was drained by penetrating diathermy at two points—one at the equator just above the upper margin of the internal rectus and the other at the equator in the lower nasal quadrant.

A retinal tear was present in 5 cases; in one of these there were three tears. In two of these cases the retina was torn by the passage of the foreign body through it. In two instances the tear and detachment were the result of blast, and in a third these were caused by the contusion from a blow with the butt end of a rifle. In two cases there was doubt as to the presence of a minute tear. In the remaining 13 cases no tear was evident.

Vitreous

There was prolapse of vitreous at the site of a penetrating wound in one case, a knuckle about 3 mm. in diameter was lost. Some vitreous haemorrhage was present in 5 cases at the time of wounding, and in one case it occurred 9½ months after an operation for retinal detachment. A moderate vitreous haemorrhage was evident in 7 cases. The contracture of organized bands of fibrous tissues was a factor in producing the detachment in 9 cases. Fig. 3

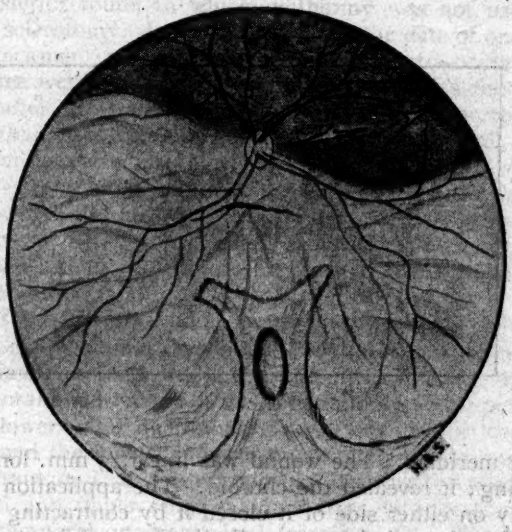

FIG. 3.

shows, like Fig. 1 the result of contracture of fibrous tissue bands in a contused eye. It is the fundus of the right and only eye of a German prisoner. He was wounded in the face with multiple shell fragments. The retina became detached 14 months after he was wounded. Operative treatment was successful in this case. A line of diathermy applications was made on the sclera beyond the limits of the scarred area, the inter-retinal fluid was drained, and the detached retina pinned down to the choroid. The retina remained in place.

Intra-ocular pressure. In 6 cases of severe contusion of the eye the intra-ocular pressure was reduced. In two of these with total detachment of the retina the intra-ocular pressure was less than 6 mm.Hg in one case and 6·5 mm.Hg in the other. In one case of severe contusion with retinal detachment on the temporal side the intra-ocular pressure was less than 6 mm.Hg. In the 4 other cases it was from 1·25 to 9·5 mm.Hg lower than that in the normal other eye. In two cases the intra-ocular pressure was higher in the eye with the retinal detachment than in the other (normal) eye. This amounted to 1·25 mm.Hg in one case and 4 mm.Hg in the other. After operation the recovery of intra-ocular pressure was slower than in the other groups of retinal detachment. The normal was not reached until 10 to 11 months after operation, and in some instances the intra-ocular pressure remained 1·5 to 3 mm.Hg below this.

Results of operation

Seventeen out of 20 cases in this group were operated on. In 11 the retina was completely replaced. Of the 6 failures, 3 were at first successful but later showed a recurrence localized to part of a quadrant of the fundus, the onset of this after operation being 6 weeks in one case, 5 months in another, and 8 months in the third case. The patient whose retinal detachment recurred 5 months after operation had for military reasons to be evacuated 18 days after operation, and the long journey from Egypt round the Cape of Good Hope to the United Kingdom may have been a factor in causing this misfortune. A spontaneous vitreous haemorrhage 8 months after operation was probably responsible for another case. In one case it was difficult to account for a shallow residual detachment in the lower part of the eye. This patient had an intra-ocular foreign body extracted by the giant electro-magnet and the detachment treated by diathermy. No definite hole was found in the retina, and despite the recurrence of the detachment his vision was 6/9 and the intra-ocular pressure within physiological limits. In another case a shallow detachment persisted in the lower part of the eye up to two months after operation, when the patient was last seen. Vitreous bands were probably the cause. In the sixth case the operation failed to seal off a large rent at the ora serrata

which occupied most of the lower temporal quadrant and to secure replacement of over half of the retina. A further operation is necessary in this case.

Vision : Visual field : Refraction

Vision improved after operation in 7 cases. In one case vision was 6/18 before operation and improved to 6/9 (partly) after operation. In another it was 6/36 before operation and was reduced to counting fingers at 1 foot as a result of a late vitreous haemorrhage 9½ months after operation. In the remaining cases the vision was made neither better nor worse by operation, whether this was successful or not.

In 7 cases the visual field was improved. In one case in which there was a vitreous haemorrhage 9½ months after operation it became 10° worse than it was before operation, and in the others there was no appreciable change. In 16 cases in this group the intra-ocular changes as a result of severe trauma made it impossible to estimate the refraction with any measure of accuracy before operation. In one case astigmatism amounting to + 1·25 axis 15° was evident after operation; but there was a posterior cortical traumatic cataract in this case, so it is difficult to be sure whether the altered refraction was the result of operation or not. In this instance the operation of pinning back a curtain-like detachment in the upper and lower half of the fundus (see Fig. 1) was successful, and the visual result was 6/9, with an unavoidable sacrifice of 30° in the temporal field. In another case the axis of the astigmatism changed from − 1·25 cyl. ax. 90° before operation to − 1·5 cyl. ax. 45° after operation.

Comparative results of operative treatment

TABLE C.

	Retinal Detachment Group	Successes	Failures
1	Trauma (Military)	11	6
2	Trauma (Civil)	23	3
3	Choroido-retinal degeneration	26 (100 per cent.)	—
4	Cystic degeneration	16 (100 per cent.)	—
5	Myopia	6	2
		82	11

I have counted as a failure any recurrence of retinal detachment however limited and slight, as indeed this was in 6 cases.

Of the 17 cases operated on in the B.L.A. there were 16 successes and 1 failure. The failure occurred in a middle-aged Yugo-Slav officer, a repatriated prisoner of war in whom three quadrants of the retina had been detached in a myopic eye for 4 years. There was a large ragged rent affecting the upper and lower temporal quadrant. Operation sealed the rent securely and the visual field became nearly full but a shallow detachment recurred 6 weeks after the operation near the ora serrata below. Further operative treatment may have to be considered later if this persists and increases.

The 76 operated on in the M.E.F. showed 86·8 per cent. successes and 13·2 per cent. failures.

It will be seen from Table C. that the results of operative treatment in the military trauma cases compare ill with the other types of detachment. An explanation of this is, I think, the gross intra-ocular damage caused to eyes by the severe contusion effects from war missiles traversing the orbit or parts of the cranium and face near the orbit. The intra-ocular pressure is considerably reduced compared with other types of retinal detachment. In severe cases no measure could be obtained with a Schiötz tonometer (it was less than the lowest reading of 6 mm.Hg) and in others less severe it remained for some months as low as 6-8 mm.Hg.

Other factors such as organizing vitreous haemorrhage and sheets of blood on the retina and between it and the choroid have already been mentioned.

Summary

The incidence of retinal detachment due to trauma from military missiles is rare. Twenty cases have been analysed. Eighteen of these occurred in the Western desert campaign 1941-43 and 2 in the invasion of Normandy, Belgium and Germany in 1944-45.

The results of treatment compare ill with other types of retinal detachment occurring in a field force.

One case was considered too hopeless to benefit by operation; 2 others underwent spontaneous recovery; and of the 17 which were operated on 11 were successes and 6 were failures. Pathological reasons for failure are discussed.

I express my thanks to all those ophthalmic colleagues in the Army, serving in the M.E.F. and the B.L.A. who were good enough to send these interesting cases to me and to Brigadier C. W. Graham, Brigadier the Honble. G. Bridgeman, M.C. and Brigadier G. I. Scott, consultants in Ophthalmology in the M.E.F. 1940-43 and to Lt.-Colonel A. Lister, R.A.M.C., Adviser in Ophthalmology, B.L.A., for their interest and advice from time to time.

ANNOTATIONS

The Faculty of Ophthalmologists

The Council of the Faculty of Ophthalmologists has issued its first Annual Report covering its activities from April, 1945, to March, 1946. The report—a lengthy document of 76 pages, comprising twenty-one sections and twelve appendices — amply demonstrates that the Faculty is playing an active part and assuming heavy responsibilities both in the academic and political aspects of ophthalmology. It is, indeed, a matter of congratulation that at the present time of revolution in all aspects of the practice of ophthalmology and re-orientation of the policy of education and research, the specialty has combined to form a single representative body through which it can express its views authoritatively. In the future its value may be still greater if ophthalmology is to survive as a living and effective science when its practice becomes bureaucratically controlled.

The main activities of the Faculty during the past year have, as would be expected, been medico-political, and for this purpose a very useful working arrangement has been entered into with the Ophthalmic Group Committee of the British Medical Association. The Negotiating Committee which makes contact with the Ministry of Health on the National Health Bill thus represents a complete political unity of the speciality and at the same time receives the backing of the entire medical profession for its views. In addition to this paramount matter the Faculty is interesting itself actively in determining the conditions for the ophthalmological care of school children with the Ministry of Education and with Industrial Ophthalmology.

With regard to more academic matters the close liaison established with the Royal College of Surgeons of England is indeed propitious and the introduction of an ophthalmological fellowship on the lines adumbrated in the report should go a long way to fulfil the need for a suitable higher diploma which it is expected will come to be recognized as the hall-mark of consulting status and the desirable qualification for appointment to a first rank Hospital Staff. It is to be hoped that similar arrangements will be made by the corresponding collegiate bodies in Scotland. Ophthalmic research, ophthalmic literature and the international contacts of British ophthalmology have all their place in the Report, as also have subjects such as the notification of trachoma and the definitions of blindness on which a detailed and reasoned memorandum appears. The Faculty also acts *in loco parentis* to the sub-specialty of orthoptics, sponsoring the Orthoptic Board, and during the past year considerable developments have taken place in its administration. It is interesting that steps are already in train for the

acceptance of similar responsibilities with regard to contact lenses on which research has been initiated.

At the end of its first year the Faculty had 469 members and associates. In the interest of ophthalmologists themselves, of the specialty which they practise, and of the community which they serve, it is to be hoped that this number will rapidly increase so that in the difficult times ahead all views in the profession may find representation and will have the most authoritative backing possible.

Drugs and their substitutes

Smollett, as a medical man, must have known what he was writing about of the practice of his day. In *Roderick Random* we have the picture of a dishonest apothecary who "was the most expert man at a succedaneum of any apothecary in London; so that I have sometimes been amazed to see him, without the least hesitation, make up a physician's prescription, though he had not in his shop one medicine mentioned in it. Oyster shells he would invent into crab's eyes; common oil into oil of sweet almonds; syrup of sugar into balsamic syrup; Thames water into aqua cinnamoni, turpentine into capivi; and a hundred more costly preparations were produced in an instant, from the cheapest and coarsest drugs of the *materia medica*."

Times have changed, and we doubt whether anyone in his senses today would take Thames water for aqua cinnamoni.

So far as we are aware there are no cheap substitutes for the common drugs we use in ophthalmic practice; but it need not be considered unreasonable to make use of common and cheap drugs for oral administration, if stocks of the more expensive drugs are low, provided the action of the two is similar. During the war several drugs were in such short supply as to be practically unobtainable and substitutes had to be found. There was nothing dishonest in this. But with the war over, we hope that shipping facilities will be available to allow a medical man to order what drugs he likes with a reasonable certainty that they will be dispensed.

Drugs have a specific action, but a bottle of medicine or an eye lotion has a psychological effect as well, especially if there be not much the matter with the patient. To cater for the latter any harmless drug can be mixed with water, if it be soluble in water, and the patient will begin to get better before the bottle is finished. It is no use, however, putting a drop of water, even if it be obtained direct from the New River, into the eye of a patient with iritis, for it will not act as a substitute for atropine.

We hope that, under State control, no petty restrictions will be placed on the profession as to the drugs we may prescribe. What a nightmare of a life it will be if we have to account for every drop of homatropine and cocaine we use!

ANNUAL CONGRESS OF THE OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM

THE 66th Annual Congress of the Ophthalmological Society of the United Kingdom was held at the Royal Society of Medicine, London, on May 30, 31, and June 1. Mr. Charles B. Goulden, O.B.E., M.D., M.Ch., the President, read an address of welcome to guests and members of the Society. The guests from abroad were :—U.S.A. Dr. Arnold Knapp (New York), Bowman Lecturer; Holland—Professor H. J. M. Weve (Utrecht), Dr. E. C. Gravemeyer (Amsterdam); France—Dr. Merigot de Treigny (Paris); Belgium—Dr. Leon Coppez (Brussels), Dr. Leon Bauwens (Antwerp), Madame van Boven (Antwerp) and Dr. Jules François (Charleroi); Switzerland—Dr. A. Franceschetti (Geneva), Dr. B. Semadeni (Davos) and Madame Noelle Chome (Lausanne); Poland—Dr. W. H. Melanowski (Warsaw); Norway—Dr. H. Gjessing (Drammen); Sweden—Dr. Gunnar von Bahr (Uppsala); Chile—Dr. Miguel Millan.

The subject of the Presidential Address was "Johannes Evangelista Purkinje" and Mr. Goulden gave a vivid and interesting biographical sketch of this great scientist of the nineteenth century.

The Bowman Lecture was given by Dr. Arnold Knapp on "The present state of the Intra-capsular Cataract Extraction" a subject on which he is an acknowledged master and enjoys a world-wide reputation. He gave a comprehensive survey of the history of intra-capsular cataract extraction to-date and with characteristic and meticulous accuracy he described and discussed points in the technique of the operation. His lecture was full of wisdom and the fruits of mature experience gained from the practice of surgery for many years. It was a pleasure to listen to his valuable teaching.

The discussion on "Ocular Disturbances Associated with Malnutrition" brought out many points of clinical and scientific interest some of these controversial. It is remarkable that the ocular changes seen in many ill-nourished prisoners of war in the far East, in particular retro-bulbar neuropathy, were not present in starved prisoners in Europe. Amblyopia coincided with beri-beri and pellagra in Harold Ridley's series. The central scotoma was up to 3° from the fixation point and had steep edges. In those who recovered, the centre of the scotoma cleared first leaving an annular paracentral scotoma. The value of B1, 2, and 6 and liver injections in the treatment of retro-bulbar retinopathy was discussed.

The openers of the discussion were Air Commodore P. C. Livingston and Mr. Harold Ridley, others taking part were Dr. Dean Smith, Dr. B. Marienstrauss, Messrs. Keith Lyle, C. D.

Shapland, R. Lindsay Rea, A. Seymour Philps, W. J. W. Ferguson, A. G. Cross, A. H. Lowther and J. D. Fraser.

The Annual Dinner of the Society was held on May 30 at the Royal College of Surgeons by kind permission of the President, Sir Alfred Webb Johnson, Bt., K.C.V.O., C.B.E., D.S.O., T.D., and the Council of the College. The setting was admirable and the speeches were enjoyed for the wit these contained. Besides the guests from abroad as mentioned above, the following were also guests of the Society:—The Rt. Hon. the Lord Moran, President of the Royal College of Physicians; Sir Alfred Webb-Johnson, Bt., President of the Royal College of Surgeons; Eardley Holland, Esq., President of the Royal College of Obstetricians and Gynaecologists; P. H. Adams, Esq., President of the Ophthalmological Section of the Royal Society of Medicine; H. S. Souttar, Esq., President of the British Medical Association; Air Marshal Andrew Grant, Director General of Medical Services, Royal Air Force; Hugh F. Powell, Esq., Master of the Worshipful Society of Apothecaries of London; and Sir Wilson Jameson, Chief Medical Officer to the Ministry of Health.

In proposing the toast of the Society Sir Alfred Webb-Johnson alluded to the history of ophthalmology and remarked that Cheselden was the first surgeon to perform iridectomy (corrected by the President of the Ophthalmological Society of the United Kingdom to "iridotomy"), and the College conferred upon Sir William Bowman the Hon. F.R.C.S. Mr. Charles Goulden replied to the toast.

In proposing the toast of the guests Sir John Parsons remarked upon the presence of all three Presidents of the Royal Colleges of Medicine, Surgery, Obstetrics and Gynaecology. He allowed his humour full rein in tilting at the "hard-baked cake of custom" in the older of the Royal Colleges which in recent years had suffered some softening from the specialities.

Dr. Arnold Knapp and Air Marshal Andrew Grant, C.B., C.B.E., K.H.S., replied. Sir Arnold Lawson, K.B.E., proposed the toast of the President to which Mr. Charles Goulden replied.

Other scientific and clinical papers were read and we hope that abstracts of these will appear in this Journal at a later date.

A Joint Clinical Meeting with the Ophthalmological Section of the Royal Society of Medicine was held on May 31, and the Annual General Meeting of the Society was on the same day.

ABSTRACTS

GLAUCOMA

Meyer, Otto (New York).—The primary cause of glaucoma. *The Eye, Ear, Nose and Throat Monthly*, December, 1945.

Meyer postulates a condition of chronic phlebitis of the jugular vein or veins as having great influence in causing primary glaucoma. He has found chronic jugular phlebitis in all cases of primary glaucoma which have come under his observation. The jugular veins, if inflamed, are sensitive to pressure and show induration. The condition is nearly always secondary to a focus of infection in the oral cavity, either dead teeth or diseased tonsils. Even a remnant of infected tonsillar tissue may remain deep to the scar left by removal of the tonsil.

In the matter of treatment, teeth and tonsils must receive attention and the best treatment for the jugular condition is the application of three or four leeches over the vein. Bleeding should be encouraged for six to eight hours. This treatment to the jugulars can be repeated at fortnightly intervals if needful, until the vein is normal and not sensitive to deep palpation.

R.R.J.

CORRESPONDENCE

LOCALISATION OF INTRA-OCULAR FOREIGN BODIES

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS,—During the last 18 months there have been a number of articles in the Journal discussing the localisation of intra-ocular F. B.'s preparatory to their removal. Whatever may be the best method in an ophthalmic hospital, it was obvious that methods such as Sweet's were out of question under service conditions. Being in charge of a Base Ophthalmic Wing during the greater part of the Italian campaign, I soon realized that any practicable method had to be one in which the actual radiology could be done, as shortly and simply as possible, by non-medical staff, the interpretation of the films being done by the ophthalmic surgeon himself. This ruled out McReynold's method, which I believe was little used in the Italian theatre although the apparatus was an issue to ophthalmic units. I found the limbal ring method by far the most accurate, Skeoch's equatorial ring giving at the best only an approximation. Accuracy in localisation became increasingly important as one's preference changed, with increasing experience of the F. B.'s met with, progressively and decidedly from the anterior to the posterior route.

The one objection to the limbal ring was the time taken for what was practically a minor operation—I found nothing short of 4 sutures really kept the ring in place concentric with the cornea.

One also had to wait for the patient's return from the X-ray room to remove it. Comburg's lens seemed the obvious solution, but I was only able to obtain one, privately, when I was later posted to Austria. Practical experience with it has convinced me that it has all the desiderata of great accuracy and extreme simplicity in use. I might add that I found the Comburg lens used as routine localising method at the University ophthalmic clinics of Graz and Vienna, and that it was the method used by the German medical service during the war.

The original Comburg contact lens was made in glass by Zeiss; their products being now unobtainable, Dixey's have at my suggestion produced them here, but in perspex, thus removing the one objection of fragility. Insertion and removal can now be safely left to a sister or house surgeon without undue rise of the surgeon's blood pressure. The specimen I saw was beautifully finished and compared very favourably with Zeiss's; the opaque markers are smaller and rivetted to the perspex, and can therefore not fall out in time. For sterilisation (heat being of course out of question, Cetavlon (of Imperial Chemicals) is advised. In the lateral view) to get the longest possible base line for finding the centre of the globe, two of the markers should be in the vertical meridian. To simplify the insertion in this respect, the makers are now attempting to make the opaque marker in the form of a limbal ring instead of the 4 separate dots.

Yours faithfully,

BERNARD GLUCK.

6, WINDSOR PLACE, CARDIFF
June 3, 1946

OBITUARY

JOHN O. W. BLAND

BLAND joined the staff of the Memorial Ophthalmic Hospital in Egypt as pathologist on October 5, 1938, from the Pathological Department of St. Bartholomew's Hospital, London. He resigned on account of ill health in the summer of 1945 and died after a long illness on May 11, 1946. He was educated at the University of Cambridge and at St. George's Hospital, and followed F. H. Stewart as pathologist at the Memorial Ophthalmic Laboratory. Their work was done in close conjunction with the former Director of the Laboratory, R. P. Wilson.

He carried out interesting and important research in the aetiology of trachoma which he reported in the *Journal of Pathology and Bacteriology*, Vol. LVI, No. 2, 1944, and in the *British Journal of Ophthalmology*, August, 1945. He showed the difficulties in the way of animal experimentation when he reported the occurrence of a spontaneous folliculosis in grivet and vervet monkeys which was frequently indistinguishable from inoculated trachoma and sufficiently

common to render both species unsuitable for trachoma investigation. Baboons also of the species Papio hamadryas were found sometimes to develop spontaneous folliculosis.

When monkeys are inoculated with human trachoma containing virus bodies inclusion and a folliculosis develops, but no inclusions can be found. When material from the infected monkey is inoculated into the conjunctiva of a non-trachomatous man the clinical signs of trachoma develop and scrapings of the conjunctiva are found to contain virus inclusions. This seemed inexplicable to Bland unless it is to be supposed that the virus element in the trachomatous monkey assumes another form.

There has been expert controversy as to the exact biological status of the trachoma virus. Bland considered that it stands in an intermediate position between the rickettsiae and the large viruses, such as psittacosis, and may possibly form a link between them. He preferred to group them with the viruses but to give them a distinctive position as the "basophilic viruses" on account of the blue staining of their initial bodies and of the matrix of their inclusions which distinguishes them from the larger typical viruses which do not possess blue initial bodies and whose inclusions are acidophilic.

NOTES

Looking Up! THIS is the title of the thirty-first Annual Report of the National Society for the Prevention of Blindness Inc., 1945. A short preface by the President, Mason M. Bigelow, states that the year covered by this report marks a transition from activities dominated by war to those inspired by peace. The Society's programme is under revaluation and a new committee has been set up to study the Society's current activities and aims and to prepare a comprehensive survey of the fields in which it can most effectually operate.

The report gives brief resumés of work done during the year on such subjects as eyes in industry, glaucoma, the partially seeing child, medical social service in eye work and vocational rehabilitation of the partially sighted. In spite of war-time conditions with restrictions on travelling and a diminished personnel the Society has managed to maintain its work in industrial, medical, social, educational and nursing aspects of sight-saving. We offer our hearty congratulations. For more than a generation the Society has been doing magnificient work in the States on sight-saving, and the results are bound to have effects on similar movements all over the world.

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The Treacher Collins Prize Essay THE Council of the Ophthalmological Society of the United Kingdom announces that the subject for the next award of the Treacher Collins Prize Essay is "Nutritional Eye Disease." Essays should be submitted to the Hon. Secretary of the Society before December 31, 1947, which is the closing date for their receipt.